QUANTITATIVE INHERITANCE OF EGG WEIGHT AND RELATED FACTORS IN THE DOMESTIC FOWL

by

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PART I - THEORY AND TECHNIQUES

(1) Introduction

The choice of egg weight in the domestic fowl as the focal point of these studies is based upon several features. It is, firstly, a characteristic of major economic importance in the field of poultry breeding. Resultantly the already extensive literature on the subject of its inheritance provides a convenient picture of present century developments in the genetical study of continuous variation. As a trait displaying marked variation due to both genetic and environmental agencies it affords a convenient model for illustrating the utility, validity, and limitations, of current biometrical methods in applied population genetics. Finally its dependence on, or association with, other important production traits in the fowl - age at sexual maturity and body weight - leads naturally to a study of the associated variations of these traits, and to a study of their individual properties.

The ultimate object of these investigations, however far from complete, is increased efficiency in the genetic control of such a system of interrelated and individually complex traits. The problems arising, though here confined to poultry breeding, have analogues in diverse fields of animal, plant and human genetics.

The current phase of population genetics in poultry is largely concerned with statistical investigations into the relative importance of genetic and environmental agencies in determining the total variation of a given quantitative trait. 'Largely concerned' is perhaps an overestimate of the actual situation; the development and general application of suitable statistical techniques has not been rapid and it is only within the past decade that large scale

analysis has been carried out within the framework of this thesis, and, at that, confined to relatively few poultry research stations in the United States.

Correlations between phenotypes are, or may be with the techniques available, subdivided into components due to genetic and environmental agencies, and selection indices and breeding programmes devised for the improvement, by selection, of individual characteristics and of overall merit. Both theoretically and practically there has been a distinct advance from earlier attempts to place the inheritance of continuously varying characteristics on a simple Mendelian basis, by the arbitrary division of frequency distributions into phenotypic classes and the designation of such as genetic. The nature of the advance is two-fold. It is recognised that complex production traits as dealt with here are, or may be, dependent on multifactorial systems of genes with individually small effects and that, even within the framework of relatively constant overall environment, the phenotypic expression of a given trait may be modified by the effects of intangible and possibly uncontrollable environmental factors peculiar to individuals. The adequacy of these concepts is discussed later in more detail. It may, however, be said that recognition of the role of environment, though much neglected in early Mendelian studies, is by no means a recent development. On the other hand, as indicated above, adequate analysis has been dependent on the slower development and popularisation of suitable biometrical techniques.

Secondly, the value of such techniques is by no means confined to problems of multifactorial inheritance. They are equally applicable, and frequently more so, to cases of simpler Mendelian inheritance in the sense of singly segregating determinants with

phenotypic expression possibly affected by environment. The writer does not dispute the axiom that complex production traits, as dealt with here, fall within the multifactorial system, though the direct demonstration of such by means of segregation and linkage tests is sparse or non-existent in poultry breeding. Such inheritance, frequently regarded as a necessary corollary of the dependence of a complex trait on numerous physiological and developmental interrelationships, may in fact be demonstrated by the lack of genetic correlation between heritable 'components'. Conversely, complex traits may be defined in the form of selection indices, discriminant functions, and the like, involving independently inherited or possibly correlated components. One may, however, query the supposition of Lerner (1950, p.26) that " ..... it is exceedingly likely that whatever major genes have a reality, they have little bearing on selection in our present-day breeds of laying fowl. Major genes must have been carried to such a high frequency in improved flocks that they contribute little to the variance in egg production. They may still be of importance as differentials between breeds but the genetic variance in egg production within breeds, and most certainly within closed flocks, must by now be dependent on genes with minor phenotypic effects."

Similarly Lerner (p.291) apparently deplores the claim of Mather (1948) that the canons of the polygenic approach are rooted in and consistent with Mendelian principles. Granted, the differences of methodology and interpretation are frequently wide but, for example, statements of heritability (the proportion of variance due to genetic differences) are equally applicable to one gene or many and the derivation of similar parameters from Mendelian prin-

ciples cannot be disputed.

As with multifactorial inheritance itself, the presence or absence of major differentials requires demonstration and some, at least, of the present findings point to a scheme of relatively simple inheritance. Thus, within a particular line of Brown Leghorns maintained at the Poultry Research Centre, Edinburgh, a major part of the variation in egg weight appears to be sex-linked, i.e. confined to one of the up to 39 pairs of chromosomes which the chicken has been estimated to possess. (Lush, 1945, p.51). On the other hand these variations are small compared to a major difference between two lines. Reciprocal crosses between lines suggest this difference to be autosomal, indicating the dependence of egg weight on genes (either as entities or groups) of major and minor effect.

There is evident need for a fuller appreciation of the techniques by which such modes of inheritance are demonstrable, in particular of those discriminating between intra- and inter-population variations. Faulty conclusions in some, at least, of the following reports have already been pointed out, but one may quote Jull's (1952) review of inheritance studies on egg weight:

"Ghigi (1948) made reciprocal matings between representatives of Gallus sonnerati and White Leghorns and secured results indicating sex-linkage. ..... Waters and Weldin (1929) and Hays (1941) reported, however, a lack of sex-linkage ... .. Olson and Knox (1946) concluded that sire and dam contribute equally in the inheritance of egg weight. Waters (1941, 1945) from investigations with inbred White Leghorns was led to the conclusion that egg weight inheritance is predominantly maternal, but this was not confirmed

that

by Hutt and Bozivich (1946) or by Hazel and Lamoreux (1947) and Lerner and Cruden (1951).

Waters and Weldin (1929), among others, were of the opinion that large egg size is dominant to small egg size. On the other hand, evidence that small egg size is dominant to large egg size was submitted by Benjamin (1920), Hurst (1921), Kopec (1924) and Hays (1929, 1937)".

One could scarcely envisage a more bewildering array of apparent contradictions or genetic diversity, a source of confusion to geneticist and practical breeder alike. Some of these reports may truly reflect wide diversity of genetic make-up and the relevance of a genetic analysis only to the particular material used. It is, however, apparent that the application and extension of current techniques serves to emphasize the confusion of early investigations and provides at least some semblance of uniformity in inheritance studies. Though not specifically commented upon, evidence from at least three other sources corroborates the 'within-population' sex-linked variation of egg weight reported here.

At the same time there is a danger of uncritical applications of statistical methods, valid only on certain assumptions, leading to results of little or limited biological and practical significance. Thus whilst stress is here laid upon the utility of variance components in suggesting specific modes of inheritance, final corroboration is frequently dependent on elimination of numerous other equally likely causative factors. Examples of ambiguity are given in the interpretation of variance components in themselves equally likely to reflect maternal effects, dominance deviations, epistasis, joint operation of these and sex-linkage, and departures from the random sampling model usually assumed.

Of particular interest and importance is the validity and meaning of present modes of subdivision of genetic and environmental variance, possibly dependent on assumptions of additivity and random operation divorced from reality. In the case of age at sexual maturity discussed here it appears that different genotypes react differently to climatic variations occurring in the production year. Furthermore 'genetic variance' itself may be partly 'environmental,' and vice-versa, in the sense that genetic differences arise through acquisition by individual genotypes of individual environments, such as day-length at data of maturity. It is apparent that under such circumstances (if ever) mere statements of heritability, though useful in selection, add little to biological understanding of the genetic differences involved. From the purely pragmatic point of view it is also desirable to assess the extent of genetic variation under improved 'overall' environmental conditions. Standardisation of environment, as by elimination of climatic variation, may well result in reduction of genetic variability, either absolutely or as a proportion of the total.

The theme of these introductory remarks is developed later in full. It is first essential to trace briefly the developments of theory and practice in population genetics throughout the present century and to explain the rationale of techniques used in these studies.

The particular problems discussed concern (1) The genetic variability of egg weight with particular reference to the previously mentioned evidence of sex-linked and autosomal inheritance. The invalidity of a recent suggestion for estimation of heritability, and its bearing on these problems, is discussed.

(2) Corresponding studies on the inheritance of egg weight components, albumen, yolk, and shell. The technical work involved was carried out in collaboration with Mr. J. E. Erasmus* during his tenure of a research fellowship at the Poultry Research Centre in 1953. Techniques of measurement were under the supervision of Erasmus whilst the writer was responsible for the statistical analysis and interpretations.

Preliminary findings under item (1) have been published elsewhere, (Osborne, 1953), and subsequent findings under items (1) and (2) accepted for publication; (Osborne, 1954).

(3) Comparison of the genetic variations of egg weight with those of body weight and age at sexual maturity in regard to

(a) the prospects of selective breeding for each of these traits.

(b) their possible contributions to the sex-linked variation of egg weight and the estimation of genetic correlations.

(4) The interactions of genotype and environment introduced in age at sexual maturity by variations in date of hatch, presented in the form of a previously published report; (Osborne, 1952).

(5) Subsequent studies on the genetic and environmental variance of age at sexual maturity and their apparent dependence on 'overall' differences in environment. The concept of heritability in relation to tangible differences in environment is discussed and evidence of marked reduction of genetic variation under improved conditions exposed.

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(6) The sampling variance of heritability estimates as derived from analysis of variance, presented in the form of a previously published report; (Osborne and Patterson, 1952).

(2) Historical

2 (A) Early developments in quantitative inheritance studies

Before 1900 the prevailing theory of heredity was one of blending, supported by data on breed crosses in animals and in plant hybrids. Variation among offspring of the same parents was attributed largely to 'non-genetic' agencies - environmental effects, the inheritance of acquired characteristics, maternal impressions, telegony, and to vaguely latent ancestral heredity under the names of reversion and atavism.

Resultantly a serious objection to Darwin's theory of natural selection was the inevitably rapid reduction of genetic variation in succeeding generations. Symbolically, with blending inheritance,

$$O = \tfrac{1}{2}(P_1 + P_2)$$

where O, P_1, P_2 are the grades of offspring and parents. The corresponding relationship of variances

$$V_o = \tfrac{1}{4}(V_{P1} + V_{P2}) = \tfrac{1}{2}V_P$$

displays the halving of genetic variance expected. Darwin himself, sceptical of the efficiency of natural selection in the face of such rapidly decreasing variability, was led towards acceptance of the inheritance of acquired characteristics as resolving the difficulty.

Following the re-discovery of Mendelism in 1900 the early part of the century was characterised by the conflict between the so-called biometrical and Mendelian schools. Galton, Pearson, and Weldon had demonstrated the inheritance of continuously varying characteristics, such as human stature, by correlation studies on related individuals. They were, however, unable to reconcile such continuous variation with the particulate or quantal mode of inheritance demon-

strated by Mendel, and regarded the latter as a trivial exception to inheritance as reflected in Galton's law of ancestral heredity. Conversely, the Mendelian school, regarding all inheritance as particulate, were unable to consider continuous variation as genetic. Consequently the prevailing view was one of two kinds of inheritance, alternative or particulate for superficial characteristics, blending for continuous variation exhibited in more fundamental characteristics.

The first step was taken by Yule who pointed out that given large numbers of genes, each contributing a small amount to the expression of a given trait, Mendelian inheritance could readily give rise to the typical frequency distributions of continuous variation. Mendel himself had suggested the presence of multiple independent factors in crosses involving colour in Phaseolus. The rationale is simply an extension of the familiar 'checkerboard' model used in genetic texts to illustrate the segregation of two or three factors, and corresponds to that by which the normal distribution of mathematical statistics arises as the limiting form of binominal distribution. Thus for n genes segregating independently (or nearly so) and with equal (or nearly equal) small and supplementary contributions to phenotype, the limiting distribution corresponds to the probability generating function

$$(\tfrac{1}{2}A + \tfrac{1}{2}a)^{2n}$$

where the coefficient of A in the expansion denotes the number of 'positively' contributing alleles present in a zygote. Normal distribution arises as n tends to infinity and the contribution of each substitution, such as A for a, tends to zero. Similarly, even with few segregating factors, the contribu-

tion of environmental agencies can disturb the regularity of distinct phenotypic classes and give rise to continuous distribution. It is furthermore evident that characters showing such polygenic variation may also be subject to the effects of major genes; in the words of Mather (1949) "polygenes are the genes of fine adjustment, clothing as it were, the indispensable skeleton of major genes, and moulding the whole into the fine shape demanded by natural selection."

Nilsson-Ehle (1908, 1909) showed that continuously varying grain colour in wheat was due to the segregation of three independent, similarly acting pairs of genes. Any one factor segregating alone gave an F_2 ratio of 3 red to 1 white, two segregating together a ratio of 15; 1, and three a ratio of 65: 1, as for the well-known segregations of one, two, or three factors with dominance. Different degrees of redness appeared to be associated with the number of factors present. Dominance was not complete so that the highest degree of redness would be given by $R_1R_1R_2R_2R_3R_3$, a lower degree by $R_1R_1R_2R_2R_3r_3$, and so on throughout the series to white, $r_1r_1r_2r_2r_3r_3$. Finally the varying genetic constitution of red F_2 plants was observable in ratios of red to white in F_3 families, some consisting of 3:1, others 15: 1 and so on.

Intensity of yellow in maize was similarly investigated by East (1910). Shull (1908) observed high uniformity within lines of self-fertilised maize, with large differences between lines, and interpreted this as due to fixation within lines of genotypes originally heterozygous at many loci.

In the same year publication of the Hardy-Weinberg law illustrated the preservation of genetic variabi-

lity in randomly breeding populations. Consider a frequency array of autosomal genotypes

$$x\,AA + y\,Aa + z\,aa, \text{ where } x+y+z = 1,$$

the same distribution holding in either sex.

$$\text{Gene frequency of } A = p = x + \frac{y}{2}$$

$$\text{" " " } a = q = z + \frac{y}{2}$$

The frequency distribution of gametes is $pA + qa$ and, with random segregation and union, leads to the next generation zygotic array

$$p^2AA + 2pq\,Aa + q^2aa$$

Gametes subsequently formed have the distribution $(p^2 + pq)A + (q^2 + pq)a = pA + qa$ since $p + q = 1$, leading to constancy of zygotic array and variance in subsequent generations. With originally different distributions in the sexes one further generation is required for equilibrium.

In 1909 Johannsen published his demonstration that selection applied to a bean population with original high variability had merely sorted out 'pure-lines' and then ceased to have effect. At this relatively early stage distinction was made between genetic variation corresponding to differences between lines, and the environmental variation present between individuals of the same genotype, a distinction grievously neglected in subsequent Mendelian analysis of egg production, and its components, in poultry.

In East's experiments with ear length in maize, (East and Hayes, 1911), the mean length in F_1 of a cross between two lines lay halfway between the parental grades. The mean of F_2 was likewise intermediate between the original parents but the variance much greater than in either the parental strains or in F_1, in accordance with expected segregation. Similar variations were found in the inheritance of coro-

lla length in Nicotiana longiflora (East 1915). The means of F_3 families varied according to the F_2 plants from which they arose whilst variance in F_3 depended on the number of segregating genes. Pictorial demonstration of these features is given by Mather (1949).

Hoshino (1915) and Sax (1923) observed apparent linkage relationships between the continuously varying characteristic of seed size and the simply determined trait of seed colour in Phaseolus vulgaris. In the latter investigation average seed weight was correlated with the number of dominant alleles for colour present; it is thus evident that the correlation could be a pleiotropic effect of genes for colour, rather than linkage. This aspect was eliminated in the investigations of Rasmusson (1935) where flowering time in garden peas showed linkage with flower colour. Warren (1924) has shown that all chromosomes in Drosophila melanogaster carry polygenes affecting traits such as egg size. In Datura many traits such as plant height, area and thickness of leaves, and size of starch grains have been found to be influenced by most of the 12 pairs of chromosomes. Some of these studies are again described in detail by Mather (1949) and in many introductory texts.

Finally the 'conflict' of biometrical and Mendelian inheritance was settled in principle by Fisher (1918) and Wright (1921) who demonstrated the correlations between relatives which may exist on the supposition of multifactorial inheritance. In animal breeding the applied aspects of the problem originated largely in the demonstration by Lush (1945) of the practical value of Wright's 1921 and subsequent (e.g. 1935) publications. A more recent and com-

plete exposition within this framework, and with particular reference to poultry, is that of Lerner (1950). For these reasons the nomenclature adopted here is largely that of the American schools. Many of the statistical concepts are given in terms of Wright's (1921, 1923) method of path coefficients, readily illustrating the correlations between relatives, the subdivision of genetic and environmental variation, and the distinction between genetic and phenotypic correlation. The results are, of course, largely synoymous with those given by Fisher and by Mather, with particular reference to the section on randomly breeding populations in the latter author's Biometrical Genetics. Other synoymous results are given in the writings of Haldane and Hogben in the British schools.

Not surprisingly the impact of Fisher's and of Wright's analysis was by no means immediate in applied genetics, and in poultry breeding the early 1920's were largely characterised by the previously mentioned attempts at Mendelian analysis. A brief survey of developments in poultry genetics, as relevant to this thesis, is given in the following two sections.

2 (B) Early developments in quantitative inheritance studies in poultry.

The first attempt at Mendelian analysis in egg production was made by Pearl (1911-1915) arising out of his own and Gowell's (1902, 1903) attempts to increase performance by selection. From the results of crossing of Plymouth Rocks and Cornish birds he was led to postulate the dependence of winter production on two gene pairs. One, an autosomal dominant, was responsible for production up to 30 eggs and the other, a sex-linked dominant, for production above this figure.

Goodale (1918) and Goodale and MacMullen (1919) concluded that Pearl's theory was inadequate. Good Mendelian fits were obtainable for two autosomal genes with arbitrary subdivision at 40, 50 or 60 eggs, whilst Pearl's own data was found to agree with the same autosomal model.

These authors, and Goodale and Sanborn (1922), pointed out that a large array of component factors may contribute to the annual record. Five factors were postulated by Goodale and Sanborn as of primary importance:

(1) Age at sexual maturity.
(2) Rate of laying.
(3) Broodiness.
(4) Pauses in production, especially winter pause.
(5) Persistency, or the time of cessation of production.

Likewise Hurst (1925) devised a system of component factors and a scheme of their Mendelian analysis, whilst Hays (1924-25 and later) was led to an extensive analysis of Goodale's components. A set

of eight gene pairs was ultimately presumed to account for the entire variation in first year egg production. Similar findings for the trait of egg weight have already been mentioned and rest likewise on the arbitrary subdivision of continuous distributions.

Little verification of these suggested schemes has been forthcoming but a great deal of information has been accumulated, on the phenotypic level, of the statistical relationships between Goodale's components. Any criticism lies not upon the utility and validity of subdivision of the production cycle into components, but upon the designation of such components as simple Mendelian characteristics. Similarly confusion has inevitably arisen by the lack of distinction between genetic and phenotypic correlation.

Munro (1936) appears to be the first author to clearly recognise the fundamental distinction between phenotype and genotype made by Johannsen in 1909. His approach is not radically different from that of present-day investigators, the major difference lying in the emphasis on genetic variation by the latter and on its relative unimportance by Munro. In the opinion of the writer some of his suggestions have been over-neglected in the current phase of statistical analysis. Other than in this thesis no attempt appears to have been made to explore the consequences of his suggestion that phenotypic differences arise largely by the differential reaction of different genotypes to changes occuring throughout the production cycle. He appears, incidentally, to be one of the first to apply, on a limited basis, the methods of analysis now to be described.

As a contribution to this thesis any merit in

the subsequent section lies only in the exposition of concepts and techniques already present or latent in the literature, and in the correction of minor issues. It is hoped, nevertheless, that the section may provide a useful reference for future work in these fields as an intermediate between the purely mathematical approach, spread over numerous publications, and the mere presentation of appropriate formulae.

(3) Current Methods of Population Genetics in Poultry

3(A) The method of path coefficients

Consider a variable z determined linearly and completely by 'independent' variables (possibly correlated) x and y, the regression equation being

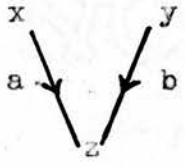
$$z = b_1 x + b_2 y$$

where variables are measured as deviations from means. Using standard measure for each variable

$$\frac{z}{s_z} = \left\{\frac{b_1 s_x}{s_z}\right\}\frac{x}{s_x} + \left\{\frac{b_2 s_y}{s_z}\right\}\frac{y}{s_y}$$

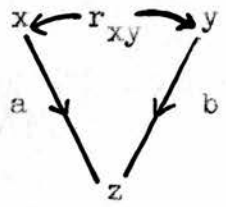
where the quantities s denote standard deviations. (Symbolism is chosen to minimise difficulties in typing encountered with the use of σ and like parameters). The quantities in brackets are respectively the standardised multiple regression coefficients of z on x and on y, i.e. the 'b-primes' as used by Snedecor (1946) in calculating the multiple regression equation. Their appellation as path-coefficients is evident from the following; denoting the coefficients by a and b respectively the system may be depicted as

(1)



with independent x and y.

or (2)



with correlated x and y.

In case (1) $r_{xz} = \dfrac{\mathrm{cov}(x, b_1x + b_2y)}{s_x s_z} = \dfrac{b_1 s^2_x}{s_x s_z} = a$

$$r_{xy} = b$$

i.e. the path coefficients and correlation coefficients are equal.

In case (2) $$r_{xz} = \frac{b_1 s^2_x + b_2 r_{xy} s_x s_y}{s_x s_z}$$

$$= b_1 \frac{s_x}{s_z} + b_2 \frac{s_y}{s_z} r_{xy}$$

$$= a + b r_{xy}$$

Similarly $r_{yz} = b + a r_{xy}$

Again in case (1)

$$s^2_z = b_1^2 s^2_x + b_2^2 s^2_y$$

and the condition of complete determination is expressed by

$$a^2 + b^2 = 1$$

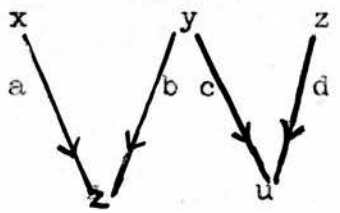
In case (2)

$$s^2_z = b_1^2 s^2_x + b_2^2 s^2_y + 2 b_1 b_2 s_x s_y r_{xy}$$

$$\text{and } a^2 + b^2 + 2 r_{xy} ab = 1$$

The method can readily be extended to more complex systems. Three examples are given below

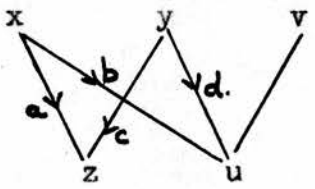
(a)



$$z = b_1 x + b_2 y \qquad u = b_3 y + b_4 z$$

$$r_{zu} = \frac{b_2 b_3 s^2_y}{s_z s_u} = bc$$

(b)

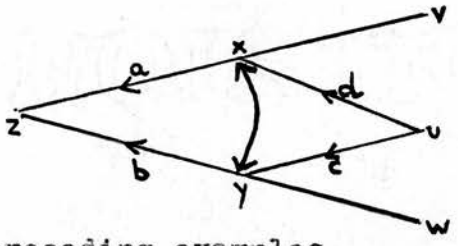


$$z = b_1x + b_2y \qquad u = b_3x + b_4y + b_5v$$

$$r_{zu} = \frac{b_1b_3s^2_x + b_2b_4s^2_y}{s_z s_u}$$

$$= ab + cd$$

(c)



From the preceding examples

$$r_{xy} = cd$$

$$r_{zx} = a + br_{xy}$$

$$= a + bcd$$

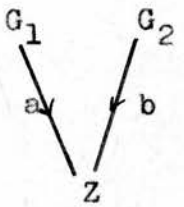
whilst the method of examples (a) and (b) gives

$$r_{zu} = ad + bc$$

The general rule in tracing paths is that, for single connecting paths, correlation and path coefficients are equal. Otherwise the correlation between two variables is a sum of products of coefficients along all paths by which the variables are connected. Along any path one, (but not more than one) connecting link may be a correlation coefficient. Paths may be traced backward (against the arrow) and then forward, or wholly forward, but never forward and then back. Any variable may be involved in more than one path but cannot be passed through twice in the same path.

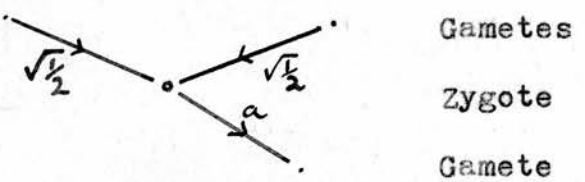
3(B) Correlations between relatives for additive autosomal inheritance.

Consider the determination of zygote by gametes with independent and additive contribution under random segregation and union.

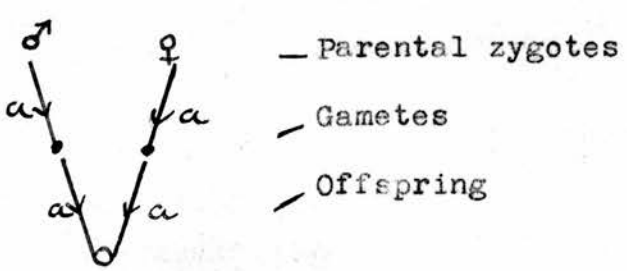


$z = G_1 + G_2$ where G_1, G_2 represent the contributions of gametes such as A, a, to zygote, the heterozygote Aa being mid-way between aa and AA in phenotypic expression. Likewise the system may represent the contributions to total genotype by any number of loci with no interaction between loci, or between alleles at the same locus. Contributions of environment are so far excluded. As before, $a^2 + b^2 = 1$ and, with equal variance of parental gametes, $a = b = \sqrt{\frac{1}{2}}$.

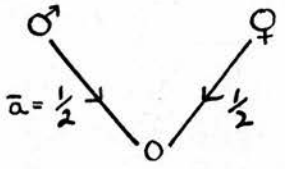
Consider next the determination by zygote of gametes produced.



Since there is only one connecting path a is equal to the correlation between gamete and zygote. This must be the same as that between zygote and determining gamete of the preceding generation, i.e. $a = \sqrt{\frac{1}{2}}$. Thus the determination of offspring by parents under random mating is represented by



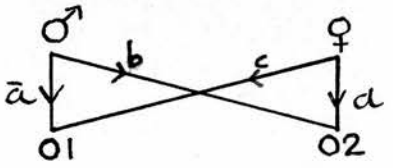
where (1) $r_{po} = a^2 = \frac{1}{2}$. The above figure may obviously be reduced to



Parents

Offspring

(2) Correlation between full-sibs

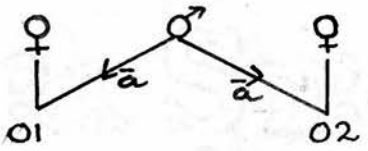


Parents

Offspring

$$r_{O1,O2} = \bar{a}b + cd = 2\bar{a}^2 = \frac{1}{2}$$

(3) Correlation between half-sibs



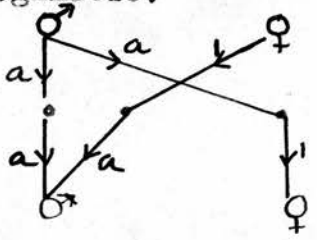
Parents

Offspring

$$r_{O1,O2} = \bar{a}^2 = \frac{1}{4}$$

3(C) <u>Correlations between relatives for additive sex-linked inheritance</u>.

Here, as relevant to poultry, the female sex is taken as heterogametic.



Parents

Gametes

Offspring

The path coefficient from female zygote to gamete or male gamete to female zygote is 1 for complete determination. As before, $a = \sqrt{\frac{1}{2}}$. Thus the correlations between offspring and parent are

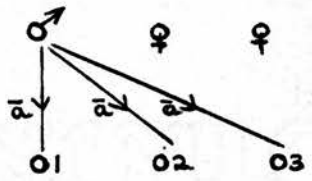
(1) Father to daughter, $a = \sqrt{\frac{1}{2}}$

(2) Father to son, $a^2 = \frac{1}{2}$

(3) Mother to daughter 0

(4) Mother to son, $a = \sqrt{\frac{1}{2}}$

Correlations between full-sibs and between half-sibs follow immediately, the cases of interest here being full-sisters and paternal half-sisters, as common in poultry breeding. In each case there is zero path from the mother and $\bar{a} = \sqrt{\frac{1}{2}}$ from the father.



Parents

Daughters

(5) Full-sister correlation $r_{O1,O2} = \bar{a}^2 = \frac{1}{2}$

(6) Half-sister correlation $r_{O2,O3} = \bar{a}^2 = \frac{1}{2}$

Incorrect values of these coefficients are given by Lerner (1950), presumably by faulty deduction from a table given by Lush (1945) for males heterogametic.

3(D) Environmental Contributions

The phenotypic expression (P) of a given trait is defined by Wright (1921) as arising by the combination of effects due to genotype (G), tangible differences in environment (T), and the intangible environmental influences (K) "which are not common even to litter mates and yet appear to be responsible for much variation in early development." Differences in notation here are to avoid confusion with symbols used later. Symbolically $P = G + T + K$ where the variables are measured as <u>deviations</u> from mean phenotype due to each agency. Assuming linear and independent contributions the corresponding relationship of variances is

$$V_P = V_G + V_T + V_K .$$

h, t, and k are the path-coefficients and

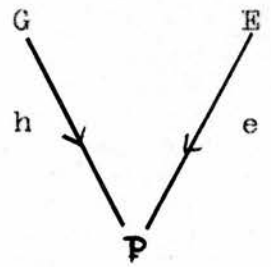
$$h^2 = \frac{V_G}{V_P} \qquad t^2 = \frac{V_T}{V_P} \qquad k^2 = \frac{V_K}{V_P}$$

the quantity h^2 denoting heritability, or the proportion of variance due to genetic differences. The situation usually assumed is that factors T and K operate at random in the population and are grouped together as a joint environmental factor E, largely beyond the control of the breeder. Major and non-randomised differences, e.g. inter-year variations, are 'eliminated' by the use of statistical techniques such as intra-year analysis of variance. Inadequacy of this view in certain cases is discussed later; meanwhile if it is assumed that phenotypic differences arise as a result of independent and additive action of differences in genotype (G) and environment (E)

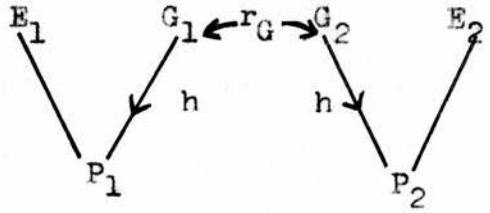
$$h^2 + e^2 = 1$$

$$\text{and } h^2 = \frac{V_G}{V_G + V_E}$$

The situation is thus depicted by



and h may be defined as the correlation between genotype and phenotype. Correlations between relatives may then be defined with the modification imposed by environment. For autosomal inheritance the coefficients of sub-section must be multiplied by h^2 as illustrated below.



P, G, and E are as defined above and the suffixes 1 and 2 refer to two individuals with genetic correlation r_G. The phenotypic correlation is $r_{P_1P_2} = r_G h^2$.

Sex linkage requires further modification. Wright (1952) gives the coefficients of sub-section 3C (p.23) multiplied by h^2, as for autosomal inheritance. It is, however, evident that heritability is different in the two sexes. For a single sex-linked gene at equilibrium under random mating the genotypic arrays are

$$♂ : p^2AA + 2pqAa + q^2aa$$

$$♀ : pA + qa$$

the genetic variance of males being twice that of females. Heritability must then be defined as h^2_m for males and h^2_f for females and correlations involving opposite sexes obtained by multiplication of the genetic coefficients by $h_m h_f$, not by h^2 as in Wright's table (unless h^2 is regarded as an average heritability). The complication does not arise when traits are expressed only in one sex, as is frequently the case in poultry genetics.

3(E) Heritability and selection

The correlation between offspring and parent under random mating and assumptions of additivity has been derived as $\frac{1}{2}h^2$. Regression coefficients, unlike correlations, are unaffected by restriction of range of the independent variable. Thus, even with selection of parents, the corresponding regression coefficient is $\frac{1}{2}h^2$, measuring heritability relative to the population from which the parents are sampled. In other words unit deviation of parental grade from mean phenotype is accompanied by deviation $\frac{1}{2}h^2$ of offspring. Thus if the mean of the two

parents deviates from population mean by an amount X, the expected offspring deviation is Xh^2, indicating the progress expected under selection. Heritability may be defined on an individual or on a family basis, providing a comparison of expected progress by different methods of selection - individual merit, family merit, or a combination of the two (Lush 1947).

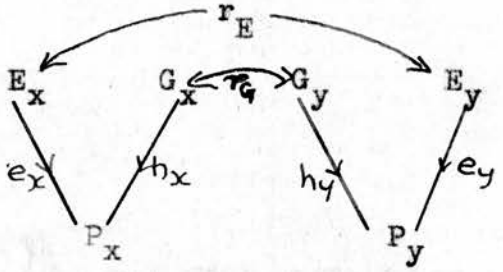
3(F) Correlations between traits.

Phenotypic correlation between two traits in the same individual may be caused by (a) the influence on each of environmental factors, (b) the influence on each of genetic constitution. The latter aspect is, of course, of primary importance in selective breeding for more than trait. Genetic correlation may be of two kinds - firstly pleiotropy, or the effect of the same genes on distinct traits, and secondly, linkage between distinct genetic determinants. Without apparent justification the latter is usually ignored in population studies as relevant to this thesis, but assumes a major part in the analysis of population variance by Mather. It is likely to be of importance in the first few generations following a cross but unlikely to be responsible for a large persisting correlation between traits in randomly breeding populations. Thus for two loci A and B the departure from random association falls off at the relative rate of c per generation, c being the average recombination frequency in ova and sperm. (Robbins 1918). Half the deviation is lost when $(1-c)^K = \frac{1}{2}$, where K is the number of generations e.g. 7 generations for 20% recombination.

It is therefore unlikely that departures from random association have serious effect on the estimation of genetic variance and correlation as in these

studies. Nevertheless elucidation of this aspect affords a future interesting problem.

Phenotypic correlations may then be depicted as below, bearing in mind the possible limitations imposed by linkage.



Here P_x is determined by the independent and additive contributions of E_x and G_x, and likewise for Y. X and Y refer to two traits in the same individual.

$$r_{P_xP_y} = h_x r_G h_y + e_x r_e e_y$$

The complexities of non-additive gene action so far preclude complete allowance for their effect on the above relationship. Their contributions to correlations between relatives are outlined in the following section.

3 (G) Non-additive effects

It is sometimes assumed that the validity and utility of heritability estimation is confined to the situation of strict additivity. Thus Woolf (1952) criticises a discriminant function as used by Mather in describing ear conformation in barley, apparently on the grounds that it presupposes strict additivity of gene action and, secondly, nearly equal contribution of each gene substitution to the character. The value of such a function, however, and of the selection indices as developed by Hazel (1943), is in maximising additive genetic variance of a combination of traits, many of which may be in fact correlated, and hence of increasing the resemblance between parent

and offspring for 'total score'.

There is, nevertheless, a great deal of confusion between techniques which estimate heritability on assumptions of additivity, and those which estimate the amount of additive variation when non-additive effects are present. The estimation of such effects is difficult in the present and comparable material from other sources, but various obscurities in the writings of Lush (1947), Hazel (1943), and others, may be mentioned.

If dominance is the only departure from additivity, total genetic variance V_G may be subdivided into a portion V_A, corresponding to the average effects of gene substitutions in the population, and a remainder V_D, due to dominance deviations. Examples of subdivision are given by Lush (1945) and by Wright (1952). Under such circumstances

$$V_P = V_G + V_E$$

$$V_A + V_D + V_E$$

and we may define the additive heritability as $h^2_A = \frac{V_A}{V_P}$. Similarly $h^2_D = \frac{V_D}{V_P}$. The correlations between relatives become

(1) Full sibs, $\frac{1}{2}h^2_A + \frac{1}{4}h^2_D$

(2) Half-sibs, $\frac{1}{4}h^2_A$

(3) Parent-offspring, $\frac{1}{2}h^2_A$

The important coefficient for selective breeding is the parent-offspring regression given by $\frac{1}{2}h^2_A$, the expected increase by selection being Xh^2_A.

h^2_A may be similarly defined if epistatic variations are present. In this case

$$V_G = V_A + V_D + V_I \text{ (I denoting epistasis)}$$

$$h^2_A = \frac{V_A}{V_P} \qquad h^2_D = \frac{V_D}{V_P} \quad \text{and} \quad h^2_I = \frac{V_I}{V_P}.$$

As in the case of dominance, differences arise between correlations of various relatives, and their magnitude depends markedly on the types of deviations operative. Thus for two gene segregations with epistasis, and with dominance at each locus, gene frequencies p_a and p_b, the correlation between offspring and parent is

$$r_{op} = \tfrac{1}{2}h^2_A + \left(\frac{p_a}{1+p_a}\right)\left(\frac{p_b}{1+p_b}\right)h^2_I$$

and that between full-sibs

$$\tfrac{1}{2}h^2_A + \tfrac{1}{4}h^2_D + \tfrac{1}{16}\left(\frac{1+3p_a}{1+p_a}\right)\left(\frac{1+3p_b}{1+p_b}\right)h^2_I$$

The general form of correlation, independent of the type of epistasis and dominance is

$$r = ah^2_A + bh^2_D + ch^2_I$$

where b is the correlation between dominance deviations of related individuals and c that between epistatic variations (c.f. Fisher 1918, Wright 1935). It is evident that only limited value can be attached to analysis of variance detection of maternal effects, as mentioned later, by comparison of half and full-sib correlations; they cannot in general be distinguished from dominance or epistasis, nor can the two be distinguished from each other.

Secondly phenotypic expression is frequently defined, and legitimately, as $P = G + E$ (deviations being implied) where G is the additive genetic value and E includes the contributions of environment, additive and non-additive, and of non-additive genetic effects (e.g. Hazel, 1943). Heritability is defined as $h^2 = \frac{V_G}{V_P}$ but the above discussion

emphasises the distinction which must be made between different estimates. Additive heritability is not in general estimated; furthermore the expected increase with selection is not necessarily Xh^2_A. The regression of offspring on mid-parent is given by $h^2_A + 2ch^2_I$. ($b = 0$ since there is nc correlation between dominance deviations of parent and offspring), i.e. the expected increase includes a contribution by epistasis.

Non-additive interactions of genotype and environment are of widespread occurrence in formal genetics but have received little attention in animal breeding. Their apparent importance on age at sexual maturity is discussed more fully in later sections.

3 (H) Analysis of variance and the estimation of heritability.

The nature of the breeding system in poultry, each sire mated to several dams, and each dam producing several offspring, renders analysis of variance of value in measuring correlations between relatives. The statistical model is that of Winsor and Clarke (1940) given in Table I.

Table I. Analysis of variance model for estimation of heritability

Source of Variation	Expected Mean Square
Between sires	$Q + nD + mS$
Between dams within sires	$Q + nD$
Between full-sibs	Q

n and m are the numbers of offspring per dam and per sire respectively. Q is the component of variance between full-sibs, D the variance between true means of dam families within sire groups, and S the variance between true means of sire families. In terms

of intra-class correlation (c.f. Fisher 1948, p.224) $S/(Q + D + S)$ is an estimate of the correlation between half-sibs and $(D + S)/(Q + D + S)$ an estimate of that between full-sibs. Estimates of heritability with additivity are thus

$\frac{S}{Q + D + S} = \frac{1}{4}h^2$, $\frac{D + S}{Q + D + S} = \frac{1}{2}h^2$ and, by subtraction, $\frac{D}{Q + D + S} = \frac{1}{4}h^2$.

A recent suggestion by Garber and Godbey (1952) has a direct bearing on the present thesis and it is eseential to point out the invalidity of their suggested correction to the above estimates. The magnitude of **S**, and hence the ratio $S/(Q + D + S)$, is magnified by sex-linkage; the resemblance between male parent and female offspring causes relative constancy between offspring in a sire group and large differences between groups. Garber and Godbey claim that the ratio is, in fact, the correlation of members of a sire group, i.e. a mixture of full and half sibs. The appropriate relationship is, they claim $S/(Q + D + S) = rh^2$ where r, greater than $\frac{1}{4}$, is the average relationship between sire's progeny. This objection is overruled by the previous estimation of individual components of variance and correction would only be necessary were analysis carried out on the restricted basis of 'between and within sires'. $(Q + D)$ estimates the population variance of half sibs and hence $S/(Q + D + S)$ the correlation between them.

Alternatively, the <u>true</u> mean of a sire family is $\frac{M}{2} + K$ where M is the contribution of the sire and K, the contribution of the dams, is, on the assumption of random sampling, constant from group to group. S thus estimates the variance of $\frac{M}{2}$ or $\frac{V_G}{4}$. Similarly D estimates $\frac{V_G}{4}$ and, since $Q + D + S$ estimates $V_G + V_E$,

the estimates of h^2 are as given above. On Garber and Godbey's assumptions high values of $S/(Q + D + S)$ are to be expected with purely autosomal inheritance and would not necessarily denote sex-linkage.

In the opposite case non-additive effects will generally cause excess of $2(D + S)/(Q + D + S)$ over $4S/(Q + D + S)$, in other words of D over S. It is customary to regard such excess as denoting maternal effects, though most of the authors concerned - Lush, Lamoreux, and Hazel (1947); Hazel and Lamoreux(1947); Lerner and Cruden (1951); and Onishi (1954), among others, are aware of the contributions of non-additivity. Without apparent reason the latter is neglected and D written as $\frac{V_G}{2} + V_M$ where V_M is the maternal effect variance. As indicated in the previous section (p. 29), with dominance $2(D + S)/(Q + D + S)$ estimates $h^2_A + \frac{h^2_D}{2}$ and $4S/(Q + D + S)$ estimates h^2_A (the suffix D denoting dominance). Likewise the correlations of epistatic variations are greater for full-sibs, and excess of the first ratio may be due to any of these causes.

Finally, maternal effects may be masked by sex-linked differences increasing S and it is apparent that excess of D is neither a necessary or sufficient condition for their existence. On the other hand significant differences may suggest crucial experimental tests of particular hypotheses.

3 (I) Estimation of Genetic Correlation.

Analysis of covariance model analogous to Table 1 was first used by Hazel, Baker and Reinmiller (1943) for estimating genetic correlations in swine. The model is given in Table 2. Q_{AB} measures the covariance of full-sibs for two traits A and B, D_{AB} and

S_{AB} are respectively the components for means of sires and dams.

Table 2. Analysis of covariance model for estimation of genetic correlation.

Source of covariance	Expected Mean Product
Between sires	$Q_{AB} + nD_{AB} + mS_{AB}$
Between dams within sires	$Q_{AB} + nD_{AB}$
Between full-sibs	Q_{AB}

It is readily shown, as before, that S_{AB} and D_{AB} each estimate $\frac{1}{4}\text{cov}(G_A G_B)$ with additive autosomal inheritance. Genetic correlation $\frac{\text{cov}(G_A G_B)}{\sqrt{V_{G_A} \cdot V_{G_B}}}$ may thus be estimated by $\frac{S_{AB}}{\sqrt{S_A S_B}}$, $\frac{D_{AB}}{\sqrt{D_A D_B}}$ or $\frac{S_{AB} + D_{AB}}{\sqrt{(S_A+D_A)(S_B+D_B)}}$.

PART II - ANALYSIS

(1) Material

The data mainly utilised in these studies were the routine records of a flock of Brown Leghorns maintained until 1950 at the Institute of Animal Genetics, Edinburgh University, and since then at the Edinburgh Poultry Research Centre of the Agricultural Research Council. In both cases the flock was under the supervision of Dr. A. W. Greenwood.

Most of the data was obtained from one line of birds, B, which serves as a control for other inbred lines selected on the bases of individual production and morphological traits. This line, because of its size and nearest approach to conditions of random mating, proves to be the most adequate for the type of biometrical analysis already outlined. No high degree of inbreeding has been practised, or selection beyond that needed to maintain an average standard of performance in general production characteristics. Line I is more highly inbred and has been selected on the basis of intensity, or rate of production.

Pullets were reared and housed intensively in 20-bird pens in a brick house under standard conditions of management within each year, the latter feature being reflected in the high degree of determination by heredity of all characteristics studied. Daily trapnesting was carried out throughout the year, special care being taken with birds near the point of lay, both in training them to enter the nests and by palpation of individuals to reveal the presence of eggs.

In these studies age at sexual maturity refers to the age in days at which the first egg is laid. March egg weight is the mean weight of all eggs laid in that month of the pullet year, each egg being weighed to the nearest 0.5 g. Body weights, to the

nearest 25 g., were measured at approximately the same time, i.e. in spring of the pullet year when mature size had been attained.

(2) Genetic Variations of Egg Weight

Analysis of variance on the model of Table 1 (p. 31) was carried out on the individual values of mean March egg weight for pullets of line B hatched between 1945 and 1949 inclusively. The measure appears to be a satisfactory criterion of maximum egg size during the pullet year and has been adopted by several authors, whose results are compared with those found here. A change to new buildings occurred in 1950 and was accompanied, in particular, by improvement in environmental conditions, such as artificial lighting. For this reason, and the limitations imposed by the extensive tabulation and analysis, the primary analysis was restricted to these five years.

Table 3 gives values of the mean squares and the corresponding components of variance Q, D and S. In calculation of the latter the mean squares were equated to

$$M_3 = Q + n_2 D + n_3 S$$

$$M_2 = Q + n_1 D$$

$$M_1 = Q$$

where the coefficients n are the appropriate estimates for unequal numbers in the subclasses; (Ganguli 1941). D was estimated as $\frac{M_2 - M_1}{n_1}$ and S by substitution of $Q + n_2 D$ in M_3. Single asterisks denote significance at the 5% point, double asterisks 1% or higher, the tests being based on F values of successive mean squares in the heirarchy.

Table 3. Analysis of variance of March, 1945-9, egg weight

	1945	1946	1947	1948	1949
Mean Square					
$Q + n_2D + n_3S$	150.6**	35.4*	141.4**	121.1**	122.8**
$Q + n_1D$	9.8	7.4	11.9	16.6*	16.8
Q	7.6	6.2	7.7	9.4	10.2
Components of variance					
S	5.10	2.08	5.72	5.35	4.39
d.f.	2	3	2	3	2
D	0.46	0.41	0.77	2.03	0.85
d.f.	14	15	10	16	8
Q	7.58	6.24	7.70	9.44	10.19
d.f.	68	37	61	58	62

Only in 1948 does the dam mean square just reach the 5% point of significance but the sire mean square is highly significant in all years except 1946, where 5% significance is still achieved.

Analysis of this type, to reduce sampling errors, is usually given in terms of a pooled intra-year analysis of variance. Here, in spite of the small number of degrees of freedom in individual years there is a remarkable, and remarkably uniform excess of S over D, to an extent incompatible with chance sampling effects.

As previously indicated, the simplest interpretation is sex-linkage. For complete sex-linked and additive inheritance the full-sib and half-sib correlations are each $\frac{1}{2}h^2_f$,

$$\frac{S}{Q + D + S} = \frac{D + S}{Q + D + S} = \tfrac{1}{2}h^2_f$$

or $D = 0$

Consider a single sex-linked gene distribution given by

♂: $p^2AA + 2pqAa + q^2aa$. Variance = $2V_L$ = male sex-linked variance

♀: $pA + qa$. Variance = V_L = female sex-linked variance.

Mating AA males to any females produces offspring of genotype $\underline{A}$ with relative frequency p^2; Aa males produce half $\underline{A}$ and half $\underline{a}$ with joint frequency 2pq; aa males produce all $\underline{a}$ with frequency q^2. Denoting the genetic values of $\underline{A}$ and $\underline{a}$ by 1 and 0 respectively the variance of $p^2AA + 2pAa + q^2aa$ is $2V_L = 2pq$. Mean values of the sire groups of offspring are $A = 1$, $\frac{A+a}{2} = \frac{1}{2}$, $a = 0$, with frequencies p^2, 2pq, q^2, and the variance of means is $S = \frac{pq}{2} = \frac{V_L}{2}$. Similarly there is no variance between means of dams in a sire group and $D = 0$. The total female sex-linked variance being V_L, the remaining $\frac{V_L}{2}$ occurs within families, giving $Q = \frac{V_L}{2}$.

Including autosomal variance V_G and environmental variance V_E the population values of components are

$$S = \frac{V_G}{4} + \frac{V_L}{2}$$

$$D = \frac{V_G}{4}$$

$$Q = \frac{V_G}{2} + \frac{V_L}{2} + V_E$$

Non-additive effects of dominance and possibly epistasis (dependent as such on the scale of measurement) and maternal effects, operate in the reverse direction. Denoting the respective variances by V_D, V_I, and V_M we have, with autosomal inheritance

$$S = \frac{V_G}{4} + k_1 V_I$$

$$D = \frac{V_G}{4} + \frac{V_D}{4} + k_2 V_I + V_M$$

where k_2 and k_1 are derived from ~~represent~~ contributions of correlations between epistatic deviations of full-sibs and of half-sibs, ~~respectively and k_2 is greater than k_1.~~ Provided the correlation for full-sibs exceeds twice that for half-sibs k_2 will be greater than k_1 and epostasis will magnify D.

Mather (1949, ch.7) discusses the question of scale of measurement in relevance to the existence of dominance and non-additivity in the more general sense of interactions between loci, and shows how such effects may be 'eliminated' by transformation of scale. There is, however, no objection to describing the inheritance of a quantitative trait in the conventional terms of Mendelian genetics. - dominance for example as the proximity of heterozygote to one homozygote in phenotypic expression of the end trait - without any assumptions as to the nature of primary gene action or developmental processes. Cell size at a particular stage in embryonic development may correspond to a heterozygous state Aa mid-way between AA and aa, but exponential or more complex growth curves give apparent dominance at later stages; in such cases the actual scale of measurement adopted may be useful in interpreting the developmental processes involved. In the present instance, however, it must be stressed that on either an additive or on a non-additive scale the simplest interpretation of excess values of S is sex-linkage. On the other hand, transformation from an additive scale to a non-additive one, or the choice of a non-additive scale in the first place, may well mask the biologically real situation of sex-linked determination, by increasing the relative value of the D component of variance.

Various factors can be postulated to give spurious excess of S over D but their contribution as

sampling errors acting consistently in one direction is highly improbable. There is no evidence of non-randomised environment, with remotely possible exceptions in 1945 and 1947 where there is a tendency for the offspring of particular sires to be housed in one pen. On the whole pens are well randomised between and within sires and there are in either case no known treatment differences. Nevertheless the possible presence of different incidences of disease, cannibalism, or other uncontrollable factors, differing between families and hence between pens, or vice-versa, emphasises the importance of randomised environment. The question of possible determination of environment by genetic constitution is considered later with respect to age at maturity.

Random mating in the strict sense is not implied in the estimation of heritability and genetic variance, and assumptions for the use of the above model are that the sires and dams are each representative samples of a population with variance V_G - such a situation applies to inbreeding arising by random breeding within a flock of finite size. In a population split up into isolated lines by inbreeding, the total variance of a character dependent on multiple genes with additive effects may be subdivided into a within-line portion, $(I-F)V_G$, and between lines, $2FV_G$, where F is Wright's (1923) coefficient of inbreeding. Within line heritability then becomes $h^2_I = \frac{(I-F)h^2}{1 - Fh^2_R}$ where h^2_R is the value under random mating.

The actual realisation of such changes is doubtful in many cases and, apart from the obvious question of mutation, much recent work throws doubt upon the validity of assumptions on which the application of Wright's coefficient depends. Pease (1948) suggests that selection for viability and heterozygosis has been responsible for maintenance

of high variability in production traits of poultry with succeeding generations of sib-mating. Gilmour (1954) has described the continued segregation of blood group alleles in the same stock, to an extent incompatible with the expected degree of homozygosis. Similar findings have been reported by Schultz and Briles (1953) whilst Düzgünes (1950) found tendency towards selective elimination of homozygotes in poultry. Hayman and Mather (1953) have shown that even a slight bias in favour of selection of heterozygotes may markedly retard the attainment of homozygosity. Reeve and Robertson (1952) describe the reduced variation of wing length in Drosophila in crosses between inbred lines and suggest possible reductions in the sensitivity of heterozygotes to environmental influences. A similar explanation is put forward by Weber and Lörtscher (1954) for the low variations in fertility of Wallace Hy-Line cocks imported into Switzerland, and by Onishi (1954) for reduced variations in age at sexual maturity in crosses between inbred lines of poultry. Similar studies have been initiated by the writer but the outcome of these findings would not appear to vitiate the results of this section, based upon the estimation of variance at a low and constant level of inbreeding.

Over the years studied the inbreeding coefficient in line B remained constant at about 0.20 but there is no deliberate system, the rise to 0.20 since 1930 being largely due to limitations in space. Extreme cases of inbreeding could cause excess of S over D. Thus it may be shown that if brother-sister mating is started in a hitherto random bred population (variance V_G), each of a random sample of

males being mated to his full-sisters, the components of variance become $S = \frac{5}{8}V_G$ and $D = \frac{1}{8}V_G$. All but one of the ratios S/D in Table 3 exceed this extreme value of 5 though such an extremity of mating system is out of the question.

The possible contribution of such factors was investigated by comparing the relationship existing between sires with the average relationship between dams in a sire-group, taking into account full-sib relationship of $\frac{1}{2}$ and half-sib of $\frac{1}{4}$. The coefficients are approximate but little error is introduced into the relative values by neglect of lower degrees of relationship. Table 4 gives the values over the individual years and it is evident that any differences are mainly in favour of higher relationship of the males, tending if anything to give values of S less than D.

Table 4. Coefficients of relationship between sires and dams

	1945	1946	1947	1948	1949
Sires	0.083	0.166	0.083	0.119	0.042
Dams	0.081	0.051	0.098	0.075	0.041

Furthermore the results of Table 3 are but trivially affected by exclusion from the analysis of any full-sister dams within a sire group.

A final consideration, though again highly improbable as a result of sampling error, is that large differences are present in the genetic means of dams in the different sire groups. Analysis of variance was carried out between and within sire groups of the phenotypic values of the dams themselves. In one year, 1949, the between groups mean square was highly significant, in 1945 there was in-

significant variation, whilst in all remaining years there was subnormal variation, i.e. less than the error term.

It is apparent that the case for sex-linked inheritance is undoubtedly strong. In any case, as in Lerner and Cruden's data (1951), and as found by Hutt and Bozivich (1946), there is complete disagreement with the findings of Waters (1941, 1945) that the inheritance of egg weight is predominantly maternal. Fallacies in this latter analysis have been discussed by Hutt and Bozivich.

Sex-linkage in traits of incomplete heritability does not necessarily imply the proximity of offspring phenotype to that of either parent and conversely, proximity to either parent does not necessarily imply sex-linkage (except when the means of populations are compared in reciprocal crosses). In so far, however, as sex-linkage implies that pullet variation should be dependent on paternal rather than maternal variation, the relative size of correlation or regression coefficients of offspring on sire and dam should serve as a criterion. In addition all the sources of objections to analysis of variance can be minimized, but complications may arise by changes in overall environment between generations. The best comparison for these purposes would be between an intra-sire regression of offspring on dam (Lush, 1940) and an intra-dam regression of offspring on sire. The former provides good estimates of heritability in poultry, particularly in view of the fact that the relatively large number of dams mated to a sire gives a good estimate of population variance. Few dams, however, are mated to more than one sire, and such a comparison is impossible.

Table 5A gives partial correlation coefficients and standardised partial regression coefficients for

offspring on 'sire' and on dam, the negligibly small contribution of correlations between parents, and differences in variance between offspring and parents, being eliminated. The important point is that sires were scored on their mothers' performances so that sire-offspring correlations and regressions are those between paternal grandmother and offspring. Thus for example $r_{OD.P}$ is the correlation between dam and offspring with the influence of the paternal grandmother eliminated. As a comparison the analysis was repeated with dams also scored on their mothers' performances, the appropriate coefficients appearing in Table 5(B). Each offspring value was taken as the unweighted mean of the family and sire values repeated for each dam or offspring value with which they were associated, the whole five years data being treated as a single sample.

Table 5. Partial correlation and standardised regression coefficients between ancestor and offspring for March egg weight.

	Individuals	Correlation Coefficient		Regression Coefficient d.f.=77	
A	Dam and offspring	$r_{OD.P}$	0.336**	$b_{OD.P}$	0.309**
A	Sire (paternal grandmother) and offspring	$r_{OP.D}$	0.395**	$b_{OP.D}$	0.372**
B	Paternal grandmother and offspring	$r_{OP.M}$	0.431**	$b_{OP.D}$	0.431**
B	Maternal grandmother and offspring	$r_{OM.P}$	0.042	$b_{OM.P}$	0.042

D = dam; O = offspring; P = paternal grandmother; M = maternal grandmother.

The coefficients, assuming randomisation of environment, point to a high degree of dependence of offspring on paternal variation. In Table 5A both the correlation coefficient $r_{OP.D}$ and the regression coefficient $b_{OP.D}$ between paternal grandmother and offspring are actually higher than the corresponding coefficients between dam and offspring. In the repeated analysis (Table 5B), where both sires and dams are scored on their mothers' records, the evidence is overwhelmingly indicative of greater determination, through the sire, by the paternal grandmother. In this respect it must be agreed, without affecting the conclusions, that the last named coefficient of Table (5B), 0.042 , appears to be smaller than expected, even with sex-linked inheritance.

It is tempting to assess relative estimates of the amount of sex-linked and autosomal variation for comparison with analysis of variance. So far, the method of estimation by multiple regression as given above, appears to preclude any simple comparison with the analysis of variance method. The possibility of an improved statistical method is under consideration by the writer.

Though not usually commented upon, similar results may be observed from other sources. For the 1935 data in Table 3 of the report by Hutt and Bozivich (1946) one may derive approximate estimates of S = 3.2, D = 0.9, though the situation is reversed in the 1941 data. The analyses refer to the mean egg weights for White Leghorn pullets in the latter half of March. The report also contains a tabulation of means in diallel crosses which the authors utilise to display, contrary to Water's findings, the influence of the sire. Complete analysis of variance of this data would be useful in displaying

sex-linked variation, but no interpretation of their data as tabulated is possible.

Lerner and Cruden (1951) present heritability estimates of egg weight for S.C.W. Leghorns, the three measures used being start of lay, November, and April egg weight. Each was the average of either a certain number of eggs or of the eggs laid in a definite period. All sire estimates of heritability, $4S/(Q + D + S)$, are in excess of the others and calculation of S and D for the three measures respectively gives (a) 2.60, 1.75 (b) 1.45, 0.73 (c) 2.71, 0.75. (The authors have acknowledged a correction to their Table 4; the figures in the column headed $2(D + S)$ should be doubled to agree with the heritability estimates, and consequently the value of $F_{2(D + S)}$ should also be doubled). Evidence of sex-linkage was not considered significant or pursued further, although the April effect was significant.

Scheinberg, Ward, and Nordskog (1953) give heritability estimates for egg weight, albumen weight, and yolk weight in New Hampshires, Barred Rocks and White Leghorns. Two eggs were selected per week for each of twenty-five successive weeks. For the three breeds respectively the S and D components for total egg weight are (a) 5.39, 2.22 (b) 5.14, 0.22 (c) 6.55, 5.19. Similar variations are evident for albumen but not for yolk, in which there is slight evidence of excess of D over S. No observations regarding sex-linkage were made.

(3) Genetic Variations of Egg Weight Components

As previously indicated, these studies were carried out in collaboration with Mr. J. E. Erasmus.

Preliminary analysis of variance of March egg weight was carried out as before for the 1950, 1951 and 1952 hatched pullets of Line B. In 1952 two samples were analysed separately. 1952B consisted of a group subject to morning and evening lighting, giving a 14-hour 'day-length' from the end of August on, just prior to maturity. 1952A was subject to evening lighting only but the amount was increased at intervals throughout the autumn season to give approximately 12 hours 'day-length'. Components S and D are given in Table 6 where it is evident that the excess of **S** is maintained, though less markedly than in 1945-9. Both estimates are small in 1950.

Table 6. Variance components of March egg weight, 1950-52.

	1950	1951	1952A	1952B
S	0.28	3.52	5.48	4.35
d.f.	2	3	3	4
D.	0.12	2.19	0.79	2.15
d.f.	12	11	12	12

Data on total, yolk, albumen, and shell weight were collected in July 1953 for all birds still in production. A sample of three consecutive eggs was taken from each bird and the measurements made as described by Erasmus (1954) in a different context. Table 7 gives the components of variance for each trait.

Table 7. Components of variance for July egg weight and its parts.

		Total	Albumen	Yolk	Shell
1952A	S	7.52*	3.34*	0.50+	0.000
	D	3.53	1.03	0.17	0.244**
1952B	S	2.64+	1.93+	0.00	0.016+
	D	4.62	2.35	0.67	0.000

+significant at 10% point
* " " 5% "
** " " 1% "
} as judged by F tests of mean squares.

The discrepancy between samples raises three interesting problems. 1952B is the only analysis in which variation of egg weight is not predominantly due to the sire. This is not a sampling effect due to the measurement of only three eggs, nor is it due to the difference in number of birds available for this and the March analysis. When the same birds are compared the components for March are S, 3.74 and D, 2.79; for average July weight (all eggs in July) the components are 1.42 and 5.80. A large difference between March and the July 3-egg sample also characterises 1952A but the components for all July in this group are less divergent from March (4.3 and 2.3 compared with 5.5 and 0.8 in March).

Tests of significance of these differences are contingent on estimates of 'repeatability' and the sampling variance of components when the same birds and families are involved, and have not yet been made. However, recalculation of all previous samples (1945-1951) for July egg weight means reveals no exception to 'sex-linked' variation, the greatest change towards equality of components occurring in 1947 where S becomes 2.3 and D 1.2 (March values 5.7 and 0.8, but not involving all the same birds). In 1950 and 1951 the estimates of D become negative.

Thus whilst sampling variance in a group where the March evidence of sex-linkage is not particularly well marked cannot be dismissed, it is suggested that the environmental effect of extended day-length and its bearing on the rhythym of production may be responsible. Preliminary calculations show that production in the A group showed the normal rise from maturity to a peak in March; in the B group a peak rate of production in November was followed by a drop in December, a slight rise in January, a steady drop from January to March, and a rise to the original peak in July. The pattern of sex-linked variation itself may well prove to be dependent on such cycles of production and hence be dependent on overall differences in environment. It may be noted that the 1950 group, in which S is very small, was also subject to 14-hour day-length from October 27th on. In other groups only evening lighting was used, and to the least extent in 1945-9 (before the change to new buildings), where the excess of S is most marked. Blyth (1952) has illustrated the complexities of the correlation between egg weight and egg number, with negative correlation expressed only in the higher producers, but no attempt has yet been made to assess the importance of variations in egg production on these results.

Secondly the pattern of components for total, yolk, and albumen follow each other in the two samples. S is greater than D for 1952A whilst the reverse is true in 1952B. There is thus the possibility that the variation of all three measures follows the pattern set originally in the weight of yolk. This is not evident in the data of Scheinberg et al. but there is a wide contrast between their method of sampling over 25 weeks, and approximately one week here.

Finally there is the interesting behaviour of shell weights. In 1952A S is larger than D for total, yolk, and albumen whilst for shell D is larger than S and highly significant, usually interpreted as denoting maternal effects. On the other hand the situation is reversed in 1952B where S (10% significance) is greater than D for shell and less for other components. This suggests a type of epistatic variation, or physiological limit, whereby small variations in egg size are accompanied by corresponding changes in shell, but large variations in the former (e.g. between sires) are not accompanied by corresponding changes in the latter. A similar conclusion follows from the regression of shell weight on egg weight for the whole 1952 sample. The range of 21 g. in egg size was divided into subgroups of 3 g. intervals. For the population of 121 eggs the total regression ignoring subgroups was 0.0674 $\pm$ 0.008 whilst the within group regression is nearly doubled, 0.1187 $\pm$ 0.037. The difference does not quite reach the 10% level of significance as judged by analysis of heterogeneity, and it must be conceded that its validity is contingent on a choice of intervals in reasonable agreement with biological fact. Nevertheless the results, subject to further investigation, provide interesting illustrations of the ambiguity and the utility of variance components in genetic analysis.

(4) Egg Weight Differences in Reciprocal Crosses

Individual phenotypes within a population may deviate from mean because of genetic or environmental contributions. As random variables the latter cancel each other out in their contribution to population mean. Genetic means and phenotypic means are thus synonymous terms and the genetic value of an individual may be defined as the phenotypic value attained when subject to the average environment of the population.

Thus the comparison of means of populations and their crosses may well reveal genetic differences. In the absence of dominance or heterosis, sex-linked differences should result in an F_1 mean nearer to one of the parental types. Dominance or heterosis of autosomal genes may give similar effects and a difference between reciprocal crosses is necessary to reveal a tendency towards one parental genotype due to sex-linkage. In the fowl the difference should arise from genetic resemblance of cross-bred pullets to pure-breds of the paternal line, due to the genetic resemblance between male parent and female offspring.

Sex-linked variation may be present in either one or both parental populations but if a major difference between them is autosomal the means of reciprocal crosses may well be equal. Such would be the case if the frequency distributions of sex-linked genes were identical in the two populations, their means being equal with respect to sex-linked loci. Alternatively sex-linked variation within populations may be small compared with the magnitude of major autosomal differences, whilst either type of variation may be dependent on the variations of other characteristics.

Line I has been selected for intensity of production and is characterised by a marked reduction in egg size below line B. The inbreeding coefficient in 1952 was 40%. The population is small and unsuitable for the application of techniques so far used in these studies. Useful comparisons are, however, obtainable from reciprocal crosses with line B.

Table 8, taken from a report by Greenwood and Blyth (1951), gives March egg weight means for lines B and I and crosses between them in each of five years in which one or more crosses were made. BI denotes B ♀ x I ♂ and vice-versa.

Table 8. Mean egg weight and standard errors for lines B, I and crosses.

	B		IB		BI		I	
	Mean	S.E.	Mean	S.E.	Mean	S.E.	Mean	S.E.
1938	61.64	0.433	56.84	0.980			49.59	0.650
1944	58.12	0.338			57.28	0.696	49.53	0.545
1946	59.13	0.435			53.20	0.796	49.79	0.491
1947	55.86	0.474	53.37	0.360	53.23	0.643	48.38	0.606
1948	57.64	0.642	52.45	0.674	56.04	0.665	46.28	0.332
Mean	58.48		54.22		54.94		48.72	

All of the cross-bred populations are small, IB averaging 25 birds and BI 21 birds. With the exception of 1946 BI, in which two males were used, all crosses consist of 1-sire families. There is thus a great deal of variability in the annual samples; in 1944 and 1948 the BI pullets average nearly as high as the female parent line whilst there is a large difference between reciprocals in 1948. There is little reason for significance tests of such compari-

sons with the standard errors given. Single sire matings are not representative of populations and such deviations are not unexpected in small groups where the mean is dependent on the particular parents, either of which may differ markedly from their population mean genotype.

The mean values over all the years should, on the other hand, give a reasonably good picture of genetic differences, in so far as chance genetic and environmental deviations should cancel out, and in view of the large line difference involved. The pure lines average 58.48 and 48.72, mean 53.60, and the reciprocal crosses 54.94 (BI) and 54.22 (IB). These realised averages may also be compared with the predicted values when the genotypes of individual sires are assessed on their full-sister records and dams on their own performance. The predicted offspring means, as the means of parents, come to 54.35 for BI and 53.59 for IB.

Bearing in mind the approximations involved there is no evidence of sex-linkage and the results are at least consistent with the presence of major autosomal differences, in contrast to the 'sex-linked' variation within line B.

(5) Genetic Association of Egg Weight and Body Weight.

There is little doubt that a major difference of 280 g. in mature body weight between lines B and I has a direct association with the egg weight differences. Mean body weights for the groups of Table 8 are

B	I	Mean of B and I	BI	IB
1806g.	1526g.	1666g.	1695g.	1665g.

the crosses again being mid-way between the parents. Genetic association of the two traits is frequently obvious, as in particular comparisons of heavy and light breeds. Apart from the above difference between lines B and I, the ranking of all inbred lines at this Centre is fairly constant for both traits; (Blyth, 1954). Many authors have shown the existence of phenotypic correlation, though few attempts have been made at subdivision into genetic and environmental contributions. Several authors (Maw, 1935; Kaufmann, 1946, 1947) have found evidence of sex-linkage for body weight in reciprocal crosses. It is thus desirable to investigate the dependence or independence of egg weight variations in Line B on those of this obviously correlated trait.

The phenotypic correlation for the 365 available comparisons from 1945-9, computed on a within-year basis, is highly significant at 0.3125, and subsequent analysis is concerned with genetic association. Table 9 gives analysis of variance of body weight for each of the years. Significance tests are again based on F tests of successive mean squares in the heirarchy, not necessarily against "error".

Table 9. Analysis of variance of body weight, 1945-9.

	Mean Squares				
	1945	1946	1947	1948	1949
Source of Variation					
(a) Sires	6273.0**	86.6	4113.5*	832.3	1899.0
(b) Dams within sires	629.4+	824.1**	660.1**	379.9	663.9**
(c) Full-sibs	350.6	181.2	182.9	290.0	216.3
Degrees of freedom					
(a)	2	3	2	3	2
(b)	14	15	10	16	8
(c)	67	37	61	58	62

\+ Significant at 10% point
* " " 5% "
** " " 1% or higher

Only in 1945 and 1947 is there any marked excess of the sire mean squares and with the exception of these two years the D component of variance is greater than S. The pooled intra-year analysis of variance is given in Table 10.

Table 10. Intra-year analysis of variance of body weight

Source of Variation	d.f.	Mean Square	Expectation
Sires	12	2277.3**	Q+5.23D+20.94S
Dams	63	622.0**	Q+4.66D
Full-sibs	285	251.2	Q
Components of variance	Q 251.2	D 79.6	S 76.8
Heritability	$\frac{4D}{Q + D + S}$	$\frac{4S}{Q + D + S}$	$\frac{2(D + S)}{Q + D + S}$
	0.78	0.75	0.77

The D component for the whole of the data is slightly in excess of S. Hazel and Lamoreux (1947) and Lerner and Cruden (1951) have found significant 'maternal effects' for body weight at 22 weeks of age and in December of the pullet year respectively but no significance can be attached to the difference between D and S here.

The estimates of heritability, 0.78, 0.75 and 0.77, are in excellent agreement and are the highest on record, those tabulated by Jull (1952, p.368) ranging from 0.32 to 0.75 with a mean value of 0.53. The writer does not wish to detract from the value of individual estimates, or of course, from his own, but estimates of sampling variance and fiducial limits are not usually available. The method of Osborne and Patterson (1952), included in this thesis, was derived to meet this obvious need and it is evident that even fairly large sample estimates can only be regarded with circumspection. Fortunately independent estimates, such as by regression analysis, are often available, and frequently subdivision of data may reveal uniformity greater than expected on the assumptions of random sampling from normal distributions.

The estimates of 0.75 and 0.78 have sampling variances of 0.1316 and 0.0467 respectively, and the joint estimate obtained by weighting estimates by reciprocals of variance is 0.77 with standard error 0.18. (Such a procedure is adopted to obtain the variance of the weighted average as $\frac{1}{V_A} = \frac{1}{V_1} + \frac{1}{V_2}$, not to obtain trivial increase in accuracy of the average of the two closely similar means. Further-

more the procedure is approximate since there is a slight correlation between estimates).

The writer has previously concluded, (Osborne, 1953), in view of the excess of S over D for egg weight, that there is no evidence of sex-linked association between the two traits. Recent analysis shows that such a view is no longer tenable and displays the limitations of orthodox estimation of genetic correlation and variation.

Analysis of variance and covariance on the models of Tables 1 and 2 (pp. 31 & 34) was carried out on an intra-year basis for the two traits. The appropriate components are given in Table 11.

Table 11. Components of variance and covariance for egg weight and body weight, 1945-9.

Egg weight	Q_E 8.30	D_E 0.98	S_E 4.59
Body weight	Q_B 251.2	D_B 79.6	S_B 76.8
Egg weight x Body weight	Q_{EB} 12.48	D_{EB} 0.65	S_{EB} 12.15

Assuming additive sex-linked and autosomal variation the expectations of components S and D for one trait are

$$S = \tfrac{1}{4}V_G + \tfrac{1}{2}V_L$$

$$D = \tfrac{1}{4}V_G$$

and it is readily shown that the corresponding components of covariance are

$$S_{EB} = \tfrac{1}{4}\mathrm{cov}(G_E G_B) + \tfrac{1}{2}\mathrm{cov}(L_E L_B)$$

$$D_{EB} = \tfrac{1}{4}\mathrm{cov}(G_E G_B)$$

where the G's refer to autosomal and the L's to sex-linked loci. Maternal effects give

$$D = \frac{V_G}{4} + V_M$$

and $D_{EB} = \frac{1}{4}(\text{cov}G_E G_B) + \text{cov}(M_E M_B)$

where the final term represents the covariance of maternal effects on E and B. Corresponding increases in D and D_{EB} (relative to S and S_{EB}) occur with non-additive effects, with possible exceptions for epistasis as mentioned on P. 41.

In this material S_{EB} = 12.15 and D_{EB} = 0.65. Comparison of variance components alone reveals no sex-linked association but there is an apparently large covariance of sex-linked effects as estimated by $2(S_{EB} - D_{EB})$. The most feasible explanation is that sex-linked variation of both traits is present but, as suggested on p. 41, the expected excess of S_B is masked by excess of D_B due to other agencies. Sex-linkage of both traits would increase S_{EB} either as a pleiotropic or linkage effect; on the other hand D_{EB} would be unaffected by maternal effects, or similar agencies, affecting one trait alone. It is of course possible that negative correlations of such agencies - e.g. negative values of $\text{cov}(M_E M_B)$ - could decrease D_{EB} and give spurious evidence of sex-linked effects.

That the former situation is operative is further suggested by regression analysis. The intra-sire regression of offspring on dam for body weight gives h^2 = 0.45, standard error 0.15. The value is not significantly different from the analysis of variance estimate of 0.77 but such a difference may well reflect the presence of non-additive effects and sex-linkage. As indicated in Part (1) p. 29, with dominance operative the regression of offspring on parent is $\frac{1}{2}h^2_A$, i.e. it excludes the contribution of dominance deviations present in the half-sib correlation of $\frac{1}{2}h^2_A + \frac{1}{4}h^2_D$. Similarly the regression of female

offspring on dam is reduced by sex-linkage to a limiting value of zero for complete sex-linked inheritance (p. 23). These points cannot, however, be emphasised too strongly; in view of unavoidable changes in environment occurring from year to year and the relative constancy of environment associated with analysis of variance and correlations between relatives, lower estimates from regression may perhaps be expected. Most estimates available are from analysis of variance but, as a measure of the average relationship between parent and offspring, the regression estimate must be regarded as a more reliable indicator of changes expected with selection. No similar analyses from other sources are available for comparison with these results.

Lerner and Cruden (1951) find similar excess values of S_E for April egg weight and subnormal values of S_B for December body weight but no details are given of individual components of variance or covariance. They derive estimates of genetic correlation corresponding to the additive autosomal model as

$$\frac{S_{EB}}{\sqrt{S_E S_B}} = 0.508, \quad \frac{S_{EB} + D_{EB}}{\sqrt{(S_E+D_E)(S_B+D_B)}} = 0.956$$

with a phenotypic correlation of 0.410. The corresponding estimates here are 0.647, 0.433 and 0.313, but it is evident that no complete assessment of genetic and environmental contributions can be made. Thus we may have

$$S_{EB} = \tfrac{1}{4}\mathrm{cov}(G_E G_B) + \tfrac{1}{2}\mathrm{cov}(L_E L_B) = 12.15$$

$$D_{EB} = \tfrac{1}{4}\mathrm{cov}(G_E G_B) + \mathrm{cov}(M_E M_B) = 0.65$$

$$Q_{EB} = \tfrac{1}{2}\mathrm{cov}(G_E G_B) + \tfrac{1}{2}\mathrm{cov}(L_E L_B) + \mathrm{cov}(E_E E_B) = 12.48$$

where any covariance may be negative or positive according to the values of other covariances. In particular it cannot be concluded from comparison of 'genetic' correlations of 0.647 and 0.433 with the phenotypic correlation of 0.313, that environmental correlation is negative i.e. that cov($E_E E_B$) is negative.

Nonsensical results may readily arise. Thus assuming no maternal or non-additive effects, autosomal correlation may be estimated as $\frac{D_{EB}}{}$ and sex-linked correlation as $\frac{S_{EB} - D_{EB}}{(S_E - D_E)(S_B - D_B)}$ $D_E D_B$ In the latter case the near realistion of a true value of $S_B - D_B$ = 0(no sex-linkage) could readily give high or even infinite estimates of sex-linked correlation. In the present instance one may conclude that autosomal correlation is absent since $\frac{D_{EB}}{D_E D_B}$ = 0.074, but only on the assumption that maternal effects etc. are absent. Maternal effects on one trait with no maternal covariance could readily give the same result.

Woolf (1952) regards the orthodox tools of analysis of variance and covariance in biometrical genetics as a "counsel of despair". The writer does not subscribe to such a contention and these same methods are used throughout this thesis to display patterns of genetic variation, limitations in the interpretations of observed effects as above, and the nature of genetic and environmental variance and interaction. The increased efficiency and co-ordination of statistical and experimental design is doubtless a desideratum of the highest order to which, however, the present techniques form an important contribution, either in practical

breeding or in biological investigation.

The relevant conclusion to this section is that, contrary to first observations, there is an apparent association of egg weight sex-linked variations with those of body weight but no discrimination between dependent and independent variations can be made. It appears from comparison of different techniques, that non-additive effects and sex-linkage may operate jointly in the determination of body weight variations.

(6) Variations of Age at Sexual Maturity.

The following studies were not initiated with respect to the possible dependence of egg weight variations on age at sexual maturity. Neverthe-:less several authors have shown an association between these traits and, in crosses between breeds or lines, there is frequently evidence of sex-linkage. Warren (1930, 1934) displayed sex-:linked behaviour in crosses between White Leg-:horns and Rhode Island Reds. In the repeated cross reported by Greenwood and Blyth the mean values of age at maturity are B,206;I,231;BI,217; IB,202; where the second letter denotes the male parent, again suggesting sex-linkage. The difference between reciprocals was significant at the 5% level. Funk and Dempster (1934) have shown that not only is early maturity associated with small egg size at start of lay but average egg size over the year and maximum monthly egg size is similarly reduced. In view of these findings it is necessary to compare the within line variations of the two traits on the level of the preceding section, particularly since the pattern of maturity variation appears to be markedly altered with improvement of environmental conditions.

The primary purpose of these studies is to assess the relative importance of genetic and environmental agencies, and their interactions, on a trait well-known to depend markedly on seasonal climatic changes. The following publication displays the genotype-environment interactions introduced in this trait by variations in date of hatch.

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(445)

XXIII.—Sexual Maturity in Brown Leghorns. The Interactions of Genotype and Environment.*

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SYNOPSIS

In each of three years studied, the spring hatching period for a flock of Brown Leghorns has been divided into two sub-periods such that a number of matings are represented by offspring in each sub-period. Analysis of variance of age at sexual maturity reveals statistical interaction between family means and hatching period, the ranking of family means varying with date of hatch. Consequences of this are discussed with reference to current methods of correcting age at maturity for date of hatch, the method of multiple shift progeny testing, and the general question of improvement by selection in the presence of non-additive combinations of genetic and environmental effects.

Measurements of comb growth in maturing pullets reveal a seasonal retardation, possibly reflecting retardation in sexual maturity, and in line with environmental effects causing cessation of production in older birds. It is suggested that rapid changes in day length during September and October may play a large part in determining the retardations and interactions displayed, and that measurements of comb growth may be of value in recognising consistently superior genotypes for age at sexual maturity.

INTRODUCTION

IN assessing the relative extent to which genetic and environmental factors are each responsible for the total variation of a given quantitative trait, the simplest plausible assumption is that phenotypic differences are completely determined by the independent and additive action of differences in genotype and environment. The total variance σ_P^2 can then be expressed as the sum of two parts, the genetic variance σ_G^2 and the environmental variance σ_E^2. Such assumptions are invalid in the presence of certain types of interaction between the two sets of factors; the effect of a given genetic increment may differ according to the environment in which it operates, whilst similarly a given environmental factor may produce a different effect on different genotypes. The general result is that the array of differences between phenotypes, and possibly their ranking, varies with the environment; additive effects alone being operative, the constancy of such array is preserved.

The heritability of trait, defined in the restricted sense as $h^2 = \sigma_G^2/\sigma_P^2$,

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or the proportion of variance due to additive genetic effects, assumes considerable importance in animal breeding as an indication of improvement to be expected by different methods of selection. Limitations in number where larger animals are concerned usually necessitate the utilisation of data from a diversity of sources, possibly the collective results of several years, or results derived from various breeding units. On the assumption of additivity, estimates of heritability may then be derived from analyses on an intra-unit basis, thus eliminating statistically those factors which supposedly affect equally all members of a given sub-population, but are not present in all populations. Non-additive effects, or interactions in the statistical sense, may seriously invalidate such estimates of heritability which may, for instance, vary between sub-populations. In addition, serious obstacles may be raised to the recognition and selection of genotypes of supposedly superior value in a range of environments or in any one environment to which the propagated stock might be exposed. Again, limitations in number might preclude the possibility of comparing the same or similar genotypes under altered conditions; and even this raises doubts as to whether or not, in many pragmatic analyses, the matter has received the attention it deserves. Particularly is this so where the existence of such effects might be anticipated—as, for example, when the phenotypic expression of a given quantitative trait may be assumed to depend on different genetic factors, such as resistance to specific disease within certain of the sub-populations. There may, then, be no grounds for the supposition that genotype A, superior to B in one environment, will be so in a second case, or that it will be so to the same extent.

A particular case in which such interaction occurs, and is readily demonstrable, is reported by Bonnier and Hansson (1948). In a series of nutritional experiments with identical twins in cattle, one member of each twin pair was subject to one of two treatments; it was thus possible to estimate variance due to heredity and that due to treatments with a residual variance representing error plus interaction. Obtaining a further estimate of error from the differences between corresponding members of twin pairs when no treatment differences were present, the significance of interaction was clearly portrayed.

For more detailed consideration of the types of interaction which may occur, and of their importance and consequence, reference may be made to Haldane (1946), Hogben (1933), and Lerner (1950, chap. viii).

The aim of the present report is to indicate that consideration of such factors appears to be of importance with reference to age at sexual maturity in the domestic fowl, with consequent effects on egg production, because of variations introduced by date of hatch (age at sexual maturity being

measured by age at first egg). A line of attack is also suggested by refinement of which it may be possible to ascertain net merit for early or optimum maturity of individuals or families hatched in different periods.

A general picture of the dependence of age at first egg on date of hatch within the Edinburgh flock may be obtained from a report by Greenwood and Blyth (1946), presenting the combined results of eleven years' hatchings. For hatches between the beginning of March and the end of April they found a progressive increase in the mean age at first egg and a corresponding increase in variance over weekly intervals, the reduced mean and variance for an August hatched group indicating a return to the conditions pertaining for early hatches. Similar results have been found by several authors and are summarised by Hutt (1949, pp. 209–211). For spring hatches between 17th March and 12th May, Byerly and Knox (1946) found an increase of about one day in mean age at maturity for every two days by which hatching date fell later than 21st March, the increase being closely correlated with decrease in day length at the actual date of maturity. The use of artificial lighting from 5 to 8 a.m. between 15th October and 1st May was found to offset retardation, but only to a slight extent. Upp and Thompson (1927) present results for hatches spread over the entire year, showing that variation of one hour in day length at date of first egg is accompanied by a variation of about fifteen days in age at first egg, and that pullets maturing after December, when day length is increasing, do so at a progressively earlier age. (Quoted from Byerly and Knox, and from Hutt.)

Changes in variance found by Greenwood and Blyth may arise in several ways, but do not indicate an additive effect causing equal retardation in all birds hatched on the same date. Naturally late maturing pullets, when subject to greater extremities of shortening day length (and possibly other seasonal effects) than more precocious ones hatched on the same date, might be retarded to a greater extent; in such case only the size of the differences between individuals would be altered by later hatching, and their ranking would remain constant. Secondly, however, additional genetic variance might be introduced by differences in resistance to the adverse autumn effects; in the absence of high positive correlation between genotype for high resistance and that for early "normal maturity", *i.e.* maturity under some standard or optimum conditions, it is evident that interactions of the type described may occur. The results already quoted indicate that changes in day length induce an immediate effect in birds which have reached a stage of near maturity; it is thus also evident that date of hatch may, in some cases, play a further part in determining interactions by controlling the length of time for which a bird, in a sufficiently

advanced stage of maturity, is exposed to the retarding influence of late autumn as a whole, as distinct from conditions pertaining near its actual date of maturity. This possibility is mentioned by Greenwood and Blyth. In such a case, even within the normal range of spring hatches, the effect of later hatching might be to reduce age at maturity in individual birds, despite a population increase in mean.

Material, Method, and Discussion

The data utilised in the present report were obtained from the 1948, 1949, and 1950 records of the flock of Brown Leghorns at the Poultry Research Centre, Edinburgh. The flock, closed since 1931, consists of several inbred lines, and first crosses between them, selected on the bases of individual production and morphological characteristics, pullets being housed and reared intensively under similar conditions. In 1950–51 artificial lighting was used morning and evening to make a 14-hour day from 27th October to 27th March, effects of changes in natural day length thus being offset or obviated between these dates. In the two previous years, evening lighting until 6 p.m. was used from the time they were penned, but morning changes in day length were still effective.

Comparison of the influence of different hatching dates was only possible on the basis of two broad periods; in 1950 these extended from 27th March to 17th April and from 24th April to 15th May, respectively. Parents used in the analyses were each represented by offspring in both periods, equal representation in each period being fairly well realised for each dam within a sire group. Table I gives details of the numbers available in 1950, classified by sire, dam, and period, whilst Table II gives the mean value of age at first egg and the number of offspring within each sub-group. In the latter and Tables VA and VB the sire number is followed by a letter or letters, denoting the line or cross to which the progeny belongs; in Table II, sires 6 and 8 were each mated to a mixed group of dams. The behaviour of the several B line families present in each set serves to indicate that the effects portrayed do not merely correspond to line or cross differences.

To investigate the significance of interaction suggested by examination of Table II, where it is evident that the ranking of sires is different in the two periods, analysis of variance was carried out by Yates' method of "weighted squares of means"; thus, as described by Snedecor (1946, pp. 284–292), allowance was made for unequal numbers in the sub-classes and unbiased estimates of variance obtained, differences based on small numbers being weighted accordingly. Results of the analyses are given

TABLE I.—NUMBER OF INDIVIDUALS WITHIN EACH PERIOD CLASSIFIED BY SIRE AND DAM

Sire	Dam	Number of Progeny Period 1 27/3–17/4	Number of Progeny Period 2 24/4–15/5
1	1	3	3
	2	2	4
2	1	5	1
	2	1	3
	3	3	2
	4	2	3
	5	2	2
	6	2	4
3	1	4	3
	2	4	4
	3	4	4
	4	2	2
	5	4	4
	6	2	4
4	1	2	5
	2	3	3
	3	2	3
5	1	5	4
6	1	2	2
	2	2	2
	3	1	1
	4	2	2
	5	2	1
7	1	3	3
	2	3	3
	3	3	3
	4	4	3
	5	3	3
8	1	1	3
	2	1	1
	3	1	1
	4	3	2

TABLE II.—MEAN AGE AT SEXUAL MATURITY IN DAYS AND NUMBER OF PROGENY IN SUB-CLASSES (1950 HATCHES)

Sire	1(B)	2(B)	3(B)	4(L)	5(DS)	6(XR)	7(N)	8(XD)
Period 1								
Mean age . .	202	203	195	219	201	231	215	198
Number . .	5	15	20	7	5	9	16	6
Period 2								
Mean age . .	192	213	203	206	202	215	207	216
Number . .	7	15	21	11	4	8	15	7

in Table III, and clearly indicate the significance of interaction, the value of F for this mean square being beyond the 1 per cent. point. Table IV gives the results of t tests between periods for each sire, the heterogeneous approach to significance in both directions being in accordance with highly significant interaction.

For the same year it was also possible to estimate the effect of interaction on an alternative basis, namely, by the differential effect observed

on dam progenies within sires. In other years the necessary exclusion of many small families—*e.g.* dams with only one offspring in a period—gave insufficient degrees of freedom on which to base such estimates of interaction variance, especially in view of the large residual variances mentioned later. For 1950, however, analysis of variance on an intra-sire basis gave an interaction mean square of 329, based on 17 degrees of freedom, with a residual mean square (within dams within periods) of 121, based on 87 degrees of freedom. The value of F is 2·72, with a corresponding z value of 0·5003, very nearly reaching the 0·1 per cent. point of 0·5246, as derived by the formula given by Fisher and Yates (1949, p. 42).

TABLE III.—ANALYSIS OF VARIANCE OF DATA OF TABLES I AND II

Source of Variation	D.F.	M.S.	F
Sires . . .	7	1453	..
Periods . . .	1	81	..
Interaction . .	7	709	3·08
Residual . . .	155	230	..

TABLE IV.—RESULTS OF t TESTS BETWEEN MEANS OF PERIODS FOR EACH SIRE

Sire	1	2	3	4	5	6	7	8
Difference of means . .	+10	−10	−8	+13	−1	+16	+8	−18
t	1·13	1·82	1·71	1·79	..	2·18	1·47	2·14
Upper limit of probability .	0·3	0·1	0·1	0·1	..	0·05	0·20	0·05

Similar results, with one important difference, were obtained by analyses of 1948 and 1949 records, and are summarised in Tables VA and VB respectively. For 1948, the periods compared were 10th March to 8th April and 16th April to 15th May, the corresponding dates for 1949 being 21st March to 4th April and 11th April to 25th April. In each case differences in ranking and the differential effect of periods on sire groups is evident, but residual variances are so large (doubtless reflecting the different conditions of lighting in these years) that the significance of interaction is reduced. As a rough comparison, it may be pointed out that for groups of ten individuals and residual mean square of 800, based on 100 degrees of freedom, 5 per cent. significance between periods for any sire would require a difference of 25 between the means. Even so, in 1949, the value of F for interaction is significant at the 5 per cent. level (Table VC).

The possibilities put forward thus appear to be fully realised, whilst more striking results are to be expected in comparisons of less broadly defined hatching periods. (It is to be pointed out in this respect that approximate equality of residual variance within periods lends validity to the above analyses. Where large differences in variance are present,

TABLE VA.—MEAN AGE AT SEXUAL MATURITY IN DAYS AND NUMBER OF PROGENY IN SUB-CLASSES (HATCHED 1948)

Sire	1(B)	2(B)	3(B)	4(IB)	5(L)	6(N)	7(D)
Period 1							
Mean age .	216	245	206	168	239	241	204
Number .	14	8	10	3	5	11	5
Period 2							
Mean age .	200	223	219	211	244	249	203
Number .	11	6	10	4	7	10	5

TABLE VB.—MEAN AGE AT SEXUAL MATURITY IN DAYS AND NUMBER OF PROGENY IN SUB-CLASSES (HATCHED 1949)

Sire	1(B)	2(B)	3(B)	4(B)	5(L)	6(I)	7(I)	8(N)
Period 1								
Mean age .	201	229	183	178	203	236	259	223
Number . .	3	7	14	8	7	4	10	12
Period 2								
Mean age .	182	227	202	186	221	246	220	223
Number . .	5	6	14	8	8	3	7	7

TABLE VC.—ANALYSES OF VARIANCE OF DATA OF TABLES VA AND VB

Source of Variation	1948			1949		
	D.F.	M.S.	*F*	D.F.	M.S.	*F*
Interaction . .	6	1249	1·42	7	1601	2·30
Residual . .	93	880	..	107	695	..

some transformation of the variable might be desirable for the use of Analyses of Variance techniques.)

If these effects occur in other flocks, their implications are far reaching. Lerner and Taylor (1940) correct age at maturity for date of hatch by using the mean age for early hatches as a base, and adjusting the values for later hatched birds by the ratio of the average value for their respective hatch to that of the base; Hutt (1949, p. 310) suggests subtracting, for each pullet, the number of days by which the mean for her hatching group

exceeds that for early hatched birds. Presupposing as they do the constant effect of later hatching on all genotypes, neither method would be adequate under the conditions described here. Secondly, as date of hatch can hardly be described as a factor varying at random between and within families, estimates of heritability and corresponding expectations of improvement by selection would be accordingly open to suspicion where a range of hatching dates is encountered, even when derived from intra-period analyses. Finally, the method of double and triple shift progeny testing described by Hutt (1949, pp. 520–523), providing production tests for a larger number of sires in one season, would be open to error where genetic differences in sexual maturity are present. As a test of the latter alone, the limitations are even more obvious, and recognition of consistently superior genotypes would be contingent rather on repeated tests of the same individuals over the usual range of hatching dates.

In 1950, conditions of lighting were constant after 27th October, so that in so far as changes in day length prior to this are responsible for differential retardations in age at first egg, the earlier use of artificial lighting, where possible, might do much to obviate this part of the variation. Further evidence that such is the case is presented in the following section.

Comb-size Variations

Evidence of retardation in sexual development is afforded by measurements of comb size taken in 1950 on 66 pullets nearing maturity, though unfortunately the choice and number of birds was not determined by the requirements of a fuller analysis of the present topic. Fig. 1 shows the hand-smoothed curves for mean comb length and mean comb height, by weekly intervals from 12th September to 7th November, of 18 birds hatched on the same date and aged 23 weeks on 12th September (both variables being measured between the furthest extremities of the comb). Variations due to hatching date were thus eliminated for the purpose of this illustration, although the corresponding curves using all the birds were closely similar.

Although analysis of variance into components due to regression, deviations from regression, and residual variation within arrays corresponding to each date indicates that regression for each variable is not significantly different from linear, an obvious explanation of the apparent curvilinearity is to be found in terms of retarding seasonal effects. It is to be noted that retardation in comb growth does not correspond to cessation of production after its onset, as out of the 66 birds used, only two were in production by 17th October (week 6 of fig. 1), the modal date

being 10th November, outside the limits of the figure. Neither is curvilinearity due merely to the rapid increase from week 6 onwards, when most birds are nearing maturity, as evidenced by several sources. Firstly, a study of individual graphs reveals marked individual retardation; secondly, the variances of comb length and height increase and then decrease instead of showing the monotonic increase associated with continuous growth, and reflect marked differences in individual retardation; thirdly, the high correlation between length and height, namely

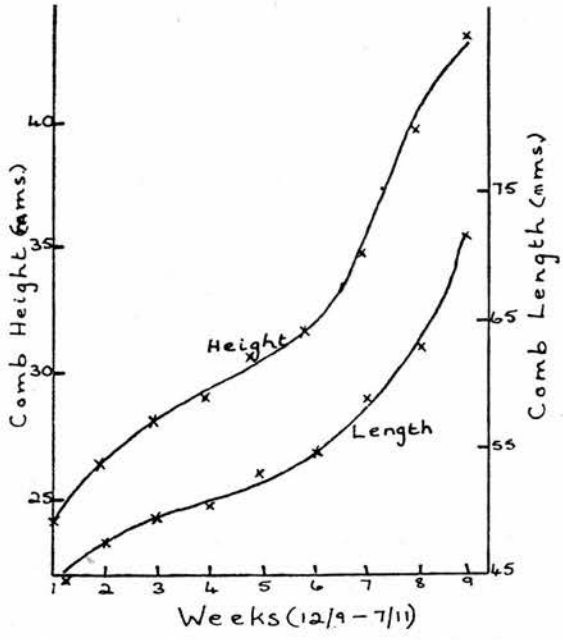

FIG. 1.—Changes in mean comb length and height for 18 birds of same age, observed between 12th September and 7th November.

0·9720 or 0·9678 with date kept constant, reflects the close correspondence of the observed effect on both variables (these correlations referring to the 18 birds of fig. 1). Finally, striking confirmation of the environmental nature of the causative factors is available from the frequency distribution for cessation of production, at the end of their laying year, of 139 birds hatched in the previous year. The cumulative frequency curve for this distribution is shown in fig. 2 as an almost exact mirror image of the curve for comb length of fig. 1.

The cumulative frequency curve corresponds to that given by Lerner and Taylor (1941) to illustrate the close association between the number of birds ceasing production and the rapidity of decrease in day length.

As, in the present case, artificial lighting was not used until 27th October (weeks 7–8 of fig. 1), the observed effects are probably traceable to similar causes, the date of greatest retardation in comb growth corresponding to the period at which day length is decreasing most rapidly. In 1950, conditions of lighting were constant from 27th October on, and it is possible that these earlier effects of shortening day length, and the different times for which birds, in a critical stage of development, are exposed to them, are largely responsible for the interactions displayed. This apart,

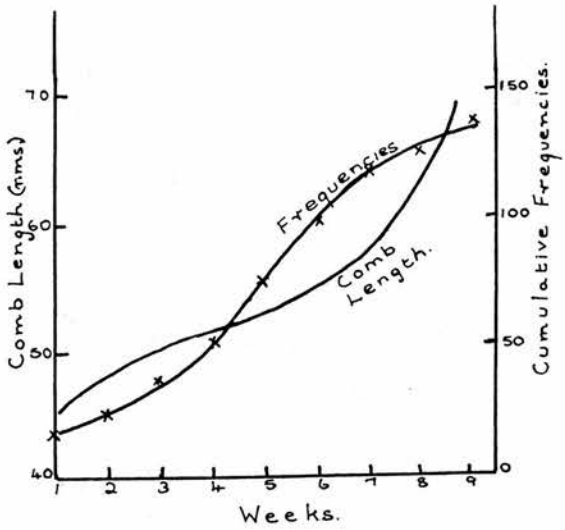

FIG. 2.—Cumulative frequency curve for cessation of production of 1949 hatched birds in juxtaposition with comb length curve of fig. 1.

it is feasible that differences in individual or family behaviour with respect to comb growth, as a manifestation of sexual development, might lead to an effective measure of retardation in age at first egg, where conditions of management preclude the possibility of eliminating the causative factors.

SUMMARY AND CONCLUSIONS

Comparison of similar genotypes (half and full sibs) under different hatching conditions supports the contention that genotype for age at sexual maturity in the domestic fowl within one hatching period cannot be identified with that within another. Due presumably to genetic differences in resistance to the adverse conditions of late autumn and the different times for which pullets, in a sufficiently advanced stage of maturity, are exposed to them, the effect of later hatching is not the same for all genotypes, and

either the ranking or the differences between phenotypes may vary with date of hatch. Consequences of this are discussed with reference to current methods of correcting age at first egg for date of hatch, the estimation of heritability and improvement by selection, and the method of double or multiple shift progeny testing.

Conditions of artificial lighting in one year, where the significance of interaction is most clearly portrayed, indicate that, apart from conditions obtaining near the actual date of maturity, rapid changes in day length in September and October may play a large part in determining the interactions displayed. Measurements of comb growth in maturing pullets support this contention, and suggest that where environmental conditions cannot be standardised, some measure of retardation in maturity might be obtainable by such methods, and recognition of consistently superior genotypes achieved.

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6(B) Relationship of Genetic Variance to Tangible Differences in Environment.

As a preliminary to constant-environment studies to be carried out at this centre it is desirable to place on record the observed variations of this trait under different conditions of artificial lighting. Limitations of 'controls' are evident here and the ultimate outcome of these findings, and their bearing on the production cycle as a whole, is dependent on experiments to be carried out by the writer in climatic chambers now under construction.

Heritability has been defined as $h^2 = V_G/V_G+V_T+V_K$ where T and K refer to tangible and intangible differences in environment. Assuming random operation of T and K the terms V_T and V_K are pooled to give a common estimate of environmental variance V_E. Again assuming independence and additivity (the effect of a given environmental factor to be the same on all genotypes and vice-versa) the variance of the population is $V_P = V_G + V_E$ and heritability $h^2 = V_G/(V_G+V_E)$.

It is difficult to envisage such a situation in regard to the climatic changes which may occur during the production cycle of the fowl. Tangible differences in environment with non-random distribution may readily be noted and a more complete understanding of the situation reached by consideration of the meaning of terms such as V_G and V_T. It is evident that such terms may be largely synonymous, in that the expression of genetic differences may be dependent on exposure to particular environmental effects.

In the specific case of age at sexual maturity discussed here, later hatched birds, or birds with slower potential rates of development, will generally complete their final stages of development under the relatively adverse conditions of late autumn. The well-known importance of changes in daylength in this context has been reviewed by Jull (1952). Alternatively, improvements in natural conditions as the season progresses may well reverse the situation. Further complications may arise by the differential reaction of different genotypes to changing conditions. The previous report illustrates changes in the ranking of family means when the same matings are represented by offspring in early and late hatches. Details are not yet available, but Abplanalp (1953), has since reported the presence of genotype-environment interactions for the same trait in the University of California flock of S.C.W. Leghorns.

It is evident that differences in <u>date</u> of sexual maturity may preclude randomisation of environment, and that phenotypic expression may be partly or largely due to the acquisition by particular genotypes of particular environments. Such a situation does not preclude breeding for age at maturity; the acquisition of environment may, assuming relative constancy of climatic variations from year to year, be regarded also as a parent-offspring relationship. It is, however, evident that statements of heritability throw little light on the nature of genetic and environmental variance. Constancy of environment may well result in reduction of V_G, V_T, V_E and of h^2, or reductions in the three variances leave h^2 unchanged.

Munro (1936) has postulated the existence of genetic differences in ability to withstand adverse effects as responsible for much of the variation in egg production. Likewise Greenwood and Blyth (1946) suggest that "the differences between genotypes (for age at sexual maturity) attain their maximum expression under the conditions when the birds approach maturity in late autumn, and that the action of genes for early and late maturity may be expressed in different degrees of refractoriness to unfavourable conditions." The obvious relationship of such reasonable assumptions to the estimation of genetic variance renders investigation as to their validity of utmost importance, both in understanding the nature of genetic variability, as opposed to an algebraic quantity, and from the purely practical side, in estimating its extent under improved conditions.

The first results refer to years 1945-9 in which the conditions of artificial lighting were limited to a maximum amount of two hours. Evening lights only were used up to 6 p.m., the onset being progressively advanced with the shortening evenings of autumn. Over the years there is a considerable range in hatching dates. They are as follows:

1945 March 27th - April 17th
1946 March 26th - May 8th
1947 April 4th - May 29th
1948 March 10th - May 15th
1949 March 14th - May 2nd

Within each year date of hatch appears to be randomised between and within families; in actual fact the variation between families is subnormal, though family differences in date of hatch may sometimes be expected due to infertility of cocks, pauses in production of the selected dams, and the emphasis placed on securing large families from particular matings.

The results of analysis of variance are given in Table 12. where levels of significance are as previously indicated.

Table 12. Estimation of heritability of age at sexual maturity, 1945-9.

Source of variation	d.f.	Mean Square	Expectation
Sires	12	4577**	Q+5.64D+23.30S
Dams within sires	64	1752**	Q+5.06D
Full-sibs	323	843	Q
Components	Q 843	D 180	S 117
Heritability	$\frac{4D}{Q+D+S}$	$\frac{2(D+S)}{Q+D+S}$	$\frac{4S}{Q+D+S}$
	0.63	0.52	0.41

Differences between the three estimates of heritability are not significant and the joint estimate, $2(D+S)/(Q+D+S)$ is 0.52 ± 0.13. When the estimates 0.63 and 0.41 are weighted by reciprocals of variance the joint estimate becomes 0.56 ± 0.06.

There is, in Table 12, an apparent excess of D over S which is also reflected in the data for the individual years as shown in Table 13.

Table 13. Components of variance for age at maturity, 1945-9.

	Q	D	S
1945	674	91	0
1946	1972	447	313
1947	602	60	53
1948	929	126	39
1949	519	112	228

Similar results from this type of analysis are given in the reports of Lerner and Cruden (1951), Onishi (1954), and in a personal communication from R. W. Hale, the Agricultural Research Institute of Northern Ireland. The significance of Onishi's results is doubtful as the population appears to be composed of a mixture of outbred stock and single-pen inbred lines. Hazel and Lamoreux (1947) found no evidence of 'maternal effects' though the D component was slightly in excess of **S.**

Two contributory factors may be mentioned with respect to the data given here. The previously computed coefficients of relationship (Table 4, p.44) are, on the average, slightly lower for dams than for sires, tending to give values of D greater than S. Secondly, the means of dams in the different sire groups are somewhat more similar than expected on the basis of random sampling. When dams are scored on their own performances analysis of variance between and within sire groups reveals the between groups mean square to be subnormal, though not significantly so. This aspect is not pursued here, the important point being the magnitude of components in Tables 12 and 13.

The recent analysis of covariance of egg weight and body weight of the preceding section has not yet been extended to age at maturity. The most that can be said, is that, in comparison of variance components, there is no obvious dependence of sex-linked variation of egg-weight on that of age at maturity. On the other hand, due to mortality between maturity and March, not all the same birds are involved in the analysis of the two traits. Recomputation of components for maturity using the same birds as in the egg weight analysis leads to the same conclusion, as shown in Table 14.

Table 14. Components of variance for age at maturity corresponding to components of egg weight.

	Q	D	S
1945	765	93	0
1946	1748	747	323
1947	604	50	41
1948	949	35	23
1949	491	81	181

The estimates of heritability appear to be the highest on record. Those tabulated by Jull (1952) have a modal value of 0.25 and range from 0.12 to 0.45. A correspondingly high estimate is also given by regression analysis, the intra-sire regression of offspring on dam giving $h^2 = 0.49$ standard error 0.13. The reduction found in the corresponding body weight estimate, possibly attributable to the joint operation of sex-linkage and non-additivity, is not apparent here. There is, however, marked variation in the yearly regression estimates which are

1945	0.40
1946	1.58
1947	0.45
1948	0.02
1949	0.64

The low value of 1948 is particularly worthy of further attention and affords example of genotype-environment interaction previously reported. Regression coefficients of age at maturity on date of hatch were computed for each family in 1948. Assuming randomisation of genotypes over the range of hatch positive regressions indicate retardation of maturity and so on. Due to small numbers the individual coefficients are subject to large sampling errors but analysis of heterogeneity reveals variation between families to be significant. The coefficients are tabulated by sire groups in Table 15, along with analysis of heterogeneity. Each coefficient corresponds to the offspring of one sire and one dam.

Table 15. Family regression coefficients of age at sexual maturity on date of hatch, 1948.

Sire	1	2	3	4
	-5.23	-0.27	0.15	0.53
	0.31	-0.29	0.18	1.54
	-3.13	1.78	0.44	1.35
	-1.49	-4.48		0.81

Analysis of heterogeneity	d.f.	MS.
Sires	3	5888 **
Dams within sires	12	1046
Pooled between families	15	2014 **
Error	47	619

** Significant at 1% point compared with error

Variance components of lighted groups

The change to new buildings in 1950 was accompanied by improved conditions in artificial lighting and its automatic control. In that year lighting was provided morning and evening to give a 14-hour 'day-length' from October 27th onwards, the effects of natural changes in day-length being offset or obviated between these dates. Hatching range was comparable to the previous years, March 20th to May 1st, but marked reduction of variance is observed. The components are Q = 352, D = 70, S = 25, giving an average estimate of $V_G = 2(D + S) = 190$ and $V_E = Q - \frac{1}{2}V_G = 257$. The corresponding average estimates for 1945-9 are $V_G = 594$, $V_E = 546$. The reduction in 'environmental' variance has been accompanied by reduction in 'genetic' variation, and a decrease in heritability, though comparison with the individual values of Table 13 reveals similarly low estimates of V_G in 1945 and 1947. On the other hand, estimates of V_G for 1948 and 1949 are 330 and 680 respectively.

Further elucidation, though limited, is obtainable in 1952 and 1953 when groups of birds of the same population were maintained by the writer under conditions of 14-hour 'day-length' from the end of August onwards. It had previously been suggested (Osborne, 1952) that the early use of artificial lighting might obviate the displayed interactions of genotype and environment. Complete control of climatic conditions was by no means achieved - artificial lights merely supplemented the normal hours of darkness, and natural changes in hours of sunlight and other vagaries of climate were fully operative.

In 1952 the difficulty of assessing changes in variability with progression of the years was counteracted by comparison of half and full-sibs in early and late hatching periods. A series of matings were each represented by offspring over a wide range of hatching dates, March 5th to May 19th. Other matings were not well represented over the whole range and their offspring were hatched primarily in the latter half of April and up to May 19th. The distribution of progeny classified by sire and dam and by two sub-periods of the hatching range is given in Table 16. The periods chosen in the first instance were March 5th-April 2nd and April 9th-May 19th, giving a maximum number of matings with offspring represented in each sub-period. Alternative classifications used later were March 5th-April 2nd, April 16th-May 19th and March 5th-April 9th.

Table 16. Distribution of progeny by sire, dam, and hatching period (1952)

Sire	Dam	Number of Offspring	
		Period (1)	Period (2)
A	a	2	1
	b	6	6
	c	4	5
	d	6	5
	e	6	3
	f	5	6
B	g	4	7
	h	2	4
	i	0	5
C	j	4	7
	k	5	8
	l	4	4
	m	2	7
	n	1	3
D	o	2	7
	p	0	3
	q	0	2
	r	0	3
E	s	4	5
	t	0	4

It will be observed that equal representation in each period is not, and in fact, cannot be achieved for all matings. Subsequent analysis was carried out firstly on the basis of all progeny, secondly progeny in the two sub-periods, and thirdly on matings represented by offspring in both periods.

For the whole data, ignoring date of hatch, analysis of variance on the usual model gave a 'joint' estimate of heritability of 0.39. The components are Q = 389, D = 55 and S = 39 on 131, 15 and 4 degrees of freedom. The range of hatch and the proportion of late hatched birds is, with the possible exception of 1948, greater than in any of the previous years but all components of variance are again reduced below the average. The formal estimates of V_G and V_E for 1945-9 are 594 and 546, for 1950 they are 190 and 257, with closely similar estimates for 1952 of 188 and 295. It is further evident that genetic variance is confined to the later hatched group, maturing later in the season. In period 1 several single matings are represented (i.e. one sire and one dam) and no attempt has been made to estimate Q, D and S for the total progeny there. However, the 'between matings' mean square is only 133 (14 d.f.), less than the error mean square of 198 (42 d.f.). The corresponding values for period 2, though involving some different matings, are 974 (18 d.f.) and 479 (74 d.f.). Period 2 as evident from Table 16, can be further analysed to give components of variance Q = 479, D = 85 and S = 76, and a joint heritability of 0.50.

Further striking differences are observed when comparison is made between matings with offspring in each period. Matings with only one offspring in a sub-period were omitted, the relevant families

being observable in Table 16. For period 1 the between matings mean square is 145 (12 d.f.) against an error term of 197 (41 d.f.); the corresponding values for period 2 are 504 (12 d.f.) and 408 (60 d.f.).

Finally the averages for the large sire families A and C are tabulated in Table 17, giving the mean age at maturity for each mating and hatching period, along with the number on which each average is based.

Table 17. Mean age at maturity for two sire groups (1952)

Sire	Dam	Period 1	Period 2
A	b	176.7(6)	176.7(6)
	c	177.8(4)	196.8(5)
	d	175.5(6)	174.2(5)
	e	169.2(6)	191.3(3)
	f	175.2(5)	172.6(5)
C	j	176.3(4)	203.6(7)
	k	177.6(5)	198.0(8)
	l	177.8(4)	190.5(4)
	m	183.5(2)	187.0(7)

As opposed to the marked variation in period 2, all but two of the means in the early group lie between 175 and 178. Analysis of variance is given in Table 18.

Table 18. Analysis of variance of data in Table 17

Source of Variation	Period 1		Period 2	
	d.f.	M.S.	d.f.	M.S.
Sires	1	109	1	2465
Dams within sires	7	43	7	482
Full-sibs	33	191	41	334

As in the data as a whole, genetic variance is absent in the early group and present in the late group. There is thus further marked evidence in the

changes of variability, of genotype environment interaction, whilst the reduction of both 'genetic' and 'environmental' variability is in line with the earlier suppositions. The observed effects are not changed by the second classification of date of hatch, March 5th - April 2nd and April 16th - May 19th. The early group is as before, whilst for all families in the late hatch heritability remains at 0.47. A further attempt was made to minimise the observed differences regarding the data of Tables 17 and 18 by omitting two birds with abnormally late maturity of 255 and 272 days. There is no very cogent reason for such procedure beyond the fact that such late individuals, occurring at low frequency, could represent deviations unassociated with date of hatch. On this basis, and on the shortened range of hatching date, the mean squares of Table 18, period 2, are changed from 2465, 482, 334 to 1551, 297 and 187. Components D and S in the former case are 26 (7 d.f.) and 83 (only 1 d.f.); in the second case they are 27 and 97, representing, in conjunction with the reduction in Q, an increase in heritability.

Similarly an extension of the early hatching range to include March 3rd - April 9th leaves the subnormality of genetic variance unchanged. As expected however, there is an increase in variance with this extension. For all progeny the between matings mean square is 253 against an error of 304 and for the data corresponding to Table 18 Period 1, the respective values of the sire, dam and full-sib mean squares become 173, 201 and 201.

A second generation was raised in 1953 under the same conditions of lighting. Dams from each of the

families in Table 17 were mated with males from the other sire group (i.e. A♂ x C♀ and C♂ x A♀) maintaining, so far as possible, the genetic variance within this group. All pullets were hatched in April, the intermediate month of 1952. Analysis of variance gives the following mean squares.

Sires	116 (3 d.f.)
Dams within Sires	238 (16 d.f.)
Full-sibs	163 (83 d.f.)

The expected mean squares $Q + nD + mS$, $Q + nD$, and Q are independent estimates of variance. Thus, comparison of the sire and error values can be interpreted as D and S equal to zero. The maximum estimate of genetic variance possible is $V_G = 4D = 56$ from a comparison of the dam and error mean squares. The corresponding estimate of V_E is 135.

If the reduction of genetic and environmental variance is real it should be reflected in greater components during the war-years when the use of artificial lighting was impossible and other conditions of husbandry, nutrition, and the like were sub-optimal. The years 1939-1942 were analysed on the preceding basis, hatching range being deliberately restricted to March and April to minimise the contributions of a few late hatched birds. 1943 and 1944 were not included, the former since the hatching season was prolonged till August, the latter since the restricted use of artificial lighting was then allowed. In actual fact, as mentioned in the previous report, the data of 1943 has been utilised by Greenwood and Blyth (1946) to illustrate changes in total variability with hatching season. For hatches between the beginning of March and end of April they found progressive increases in mean and total varia-

tion of age at maturity but the values for the late August hatched group, which matured in the following spring, were reduced to the levels obtaining for early hatches.

For 1939-1942 the components of variance are Q = 1333, D = 327 and S = 128, which again incidentally substantiate the excess of D over S. The average estimate of V_G is 910 and that of V_E is 978. The complete array of estimates of V_G and V_E is given in Table 19.

Table 19. Genetic and environmental variance of age at maturity.

	V_G	V_E
1939-42	910	978
1945-9	594	546
1950	190	257
1952	188	295
" early	0	191 or 198
" late	322	318
1953	56	135

The obvious conclusion is a reduction of genetic variance to an extent improbable on the limited inbreeding and selection present in this line. Objections on this score are not relevant for the 1952 data where offspring of the same matings are compared and where the late group shows a return to increased variability. Marked reduction in environmental variance has not been accompanied by the increase in heritability expected on normal assumptions of additivity. The extreme of improved conditions, early hatch and artificial lighting, points in fact to a value of zero within the limits of a small sample in this particular line.

For the data as a whole reduction of h^2 is not overstressed. The value for the war years remained at 0.48, closely similar to those for 1945-9. Even in 1953 regression of offspring on dam gives an estimate of 0.62 $\pm$ 0.24. This, however, includes data for early and late hatched dams of 1952. When a subdivision is made as in Table 16, regression for early dams gives an insignificant h^2 of 0.32 $\pm$ 0.48 and for late dams an impossibly high value of 1.18 $\pm$ 0.32. Sampling errors are too big to form definite conclusions but in themselves the values bear out apparent reduction of heritability with improved conditions.

Limitations of this analysis, with respect to possible natural and selective changes in variability, should be overcome in the current year by the provision of climatic chambers with complete control of lighting and temperature. Series of matings have been made to give offspring for both experimental and control groups, the latter to be maintained under the natural climatic variations of autumn and winter.

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XXIV.—On the Sampling Variance of Heritability Estimates derived from Variance Analyses.*

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Synopsis

A method is presented by which the sampling variance of all heritability estimates derived from ratios involving variance components may be found.

The statistical model of Winsor and Clarke (1940) provides a convenient method of estimating, in a population, the proportion of variance due to additive genetic effects. Thus, as described in detail by Lerner (1950, p. 120), the analysis of Table I gives for estimates of heritability,

$$\frac{h^2}{4}=\frac{B}{A+B+C}=\frac{C}{A+B+C}, \qquad \frac{h^2}{2}=\frac{B+C}{A+B+C}.$$

In the table, n and m are the numbers of individuals per dam and per sire respectively. A, B, and C are the estimates of variance components α, β, and γ associated respectively with individual, dam, and sire effects.

Table I.—Analysis of Variance Model for Estimation of Heritability

Source of Variation	D.F.	Mean Square
Between sires . . .	c	$A+nB+mC$
Between dams within sires .	b	$A+nB$
Between full sibs . . .	a	A

Few attempts have been made to evaluate the sampling variance of estimates of heritability thus derived. For restricted analyses, *e.g.* between and within sires, Robertson and Lerner (1949) derive the standard errors of their estimates as those of intra-class correlation coefficients, adapting the formula given by Fisher (1950, p. 221). From Table I, however, only the ratios $C/(A+B+C)$ and $(B+C)/(A+B+C)$ can be regarded as intra-class correlations, and even in these cases use of Fisher's standard

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error would not be strictly correct; for instance, its use corresponding to the ratio $C/(A+B+C)$ would require a "within sires" estimate of variance $A+B$; in actual fact, pooling of sums of squares in Table I gives for this mean square $A+\frac{nb}{a+b}B$.

Knapp and Nordskog (1946), for analyses between and within sires, derive the expression $h^2=4(F-1)/(K+F-1)$, K being the number of individuals within sub-classes and F the variance ratio (allowance being made here for errors in the text). By transforming values of F to z and evaluating fiducial limits for the latter, the corresponding limits for h^2 were derived. The appropriate transformation is, however,

$$z=\tfrac{1}{2}\log F=\tfrac{1}{2}\log\left\{\frac{1+(K-1)r}{1-r}\right\},$$

where $r=B/(A+B)$, A and B being the variance components involved. The sampling variance of z, for large numbers of groups (n), is

$$\frac{K}{2(K-1)(n-2)}$$

(Fisher, 1950, p. 219), so that the above would appear to be merely a roundabout way of setting fiducial limits to the intra-class correlation coefficient by transforming to z and back again, and the objections raised to regarding the ratios of Table I as intra-class correlations are again pertinent. Alternatively, if, for instance,

$$F_1=\frac{A+nB+mC}{A+nB}, \qquad F_2=\frac{A+nB}{A},$$

we have

$$nB=(F_2-1)A, \qquad mC=(F_1-1)F_2A,$$

giving

$$\frac{C}{A+B+C}=\frac{n(F_1-1)F_2}{nm+m(F_2-1)+n(F_1-1)F_2},$$

the method adopted by these authors being inapplicable to this ratio involving two values of F.

Whilst anormality of the distribution of h^2 may preclude the possibility of setting precise fiducial limits to its value, the distribution in general being closely related to that of the intra-class correlation coefficient, the method of estimating the sampling variance derived in the present report is applicable to all the afore-mentioned ratios, and provides, in a special case, a close approximation to the standard error of the latter statistic.

For continuous variables such that $z=x/y$ we have, approximately,

$$\delta z=(\bar{y}\delta x-\bar{x}\delta y)/\bar{y}^2,$$

where δx, δy, and δz are deviations from means. Thus

$$\sigma_z{}^2=[\bar{y}^2\sigma_x{}^2+\bar{x}^2\sigma_y{}^2-2\bar{x}\bar{y}\ \mathrm{cov}\ (x, y)]/\bar{y}^4. \tag{1}$$

This approximation holds provided the ratio $\sigma_y/\bar{y}$ is sufficiently small for powers higher than the first to be neglected (Kelley, 1947, p. 504). Estimates of the sampling variance of the components of Table I may be derived from the formulæ given by Crump (1946, 1951). For underlying normal distributions any mean square M, based on f degrees of freedom, is distributed (in a *balanced* classification) like $E(M)\frac{\chi^2{}_f}{f}$. The variance of χ^2 being $2f$ and the mean squares being mutually independent, we derive

$$S^2{}_A=\frac{2A^2}{a},$$

$$S^2{}_B=\frac{2}{n^2}\left\{\frac{(A+nB)^2}{b}+\frac{A^2}{a}\right\},$$

$$S^2{}_C=\frac{2}{m^2}\left\{\frac{(A+nB+mC)^2}{c}+\frac{(A+nB)^2}{b}\right\}.$$

Daniels (1939) suggests that for unbiased estimates of variance, the degrees of freedom in the above expressions should be replaced by $a+2$, $b+2$, and $c+2$ respectively. Satterthwaite, however (1941), states that such correction is not appropriate. For the case of unequal numbers in the sub-classes reference may be made to Crump (1951).

From a series of analyses involving genetic variance components in corn, Comstock and Robinson (1951) found good agreement between the estimates of sampling variance computed as above (using mean values of the mean squares) and the actual variance observed. The form of distribution of the estimates and the fiducial limits were also in agreement with expectation on the underlying assumptions.

Replacing x and y of formula (1) by, for example, C and $A+B+C$ respectively,

$$S^2{}_x=S^2{}_C,$$

$$S^2{}_y=S^2{}_A+S^2{}_B+S^2{}_C+2\{\widehat{\mathrm{cov}}\ (A, B)+\widehat{\mathrm{cov}}\ (B, C)+\widehat{\mathrm{cov}}\ (A, C)\},$$

$$\widehat{\mathrm{cov}}\ (x, y)=\widehat{\mathrm{cov}}\ (A, C)+\widehat{\mathrm{cov}}\ (B, C)+S^2{}_C,$$

where $\wedge$ denotes "estimate of". Independence of the mean squares brings in correlations between the estimates of α, β, and γ—for instance, β is estimated by substituting the value of A from the "full-sibs" mean square in that for "between dams", giving

$$\widehat{\text{cov}}\,(A,\ A+nB) = S^2_A + n\,\widehat{\text{cov}}\,(A,\ B) = 0.$$

$$\therefore\quad \widehat{\text{cov}}\,(A,\ B) = -\frac{S^2_A}{n},$$

$$\widehat{\text{cov}}\,(A,\ A+nB+mC) = m\,\widehat{\text{cov}}\,(A,\ C) = 0,$$

$$\widehat{\text{cov}}\,(A+nB,\ A+nB+mC) = S^2_A + n^2 S^2_B + 2n\,\widehat{\text{cov}}\,(A,\ B) + nm\,\widehat{\text{cov}}\,(B,\ C) = 0.$$

$$\therefore\quad \widehat{\text{cov}}\,(B,\ C) = \frac{S^2_A - n^2 S^2_B}{nm}.$$

From formula (1),

$$(A+B+C)^4 S^2_z = (A+B+C)^2 S^2_C + C^2\{S^2_A + S^2_B + S^2_C + 2\,\widehat{\text{cov}}\,(A,B) + 2\,\widehat{\text{cov}}\,(B,C)\} - 2C(A+B+C)\{\widehat{\text{cov}}\,(B,\ C) + S^2_C\}$$

$$= (A+B)^2 S^2_C + C^2\{S^2_A + S^2_B + 2\,\widehat{\text{cov}}\,(A,\ B)\} - 2C(A+B)\,\widehat{\text{cov}}\,(B,\ C).$$

Similar symmetrical relationships hold for the remaining ratios, bearing in mind that $\widehat{\text{cov}}\,(A,\ C) = 0$. Thus, for the ratio $B/(A+B+C)$,

$$(A+B+C)^4 S^2_z = (A+C)^2 S^2_B + B^2(S^2_A + S^2_C) - 2B(A+C)\{\widehat{\text{cov}}\,(A,\ B) + \widehat{\text{cov}}\,(B,\ C)\},$$

and for $(B+C)/(A+B+C)$,

$$(A+B+C)^4 S^2_z = A^2\{S^2_B + S^2_C + 2\,\widehat{\text{cov}}\,(B,\ C)\} + (B+C)^2 S^2_A - 2A(B+C)\,\widehat{\text{cov}}\,(A,\ B).$$

In each case substitution for the components, and their variances and covariances, gives the estimate of sampling variance of the ratio, from which, by multiplication by 16 or 4, the corresponding estimate for h^2 may be derived.

The formulæ for the sampling variance of z may also be obtained by the direct use of the mean squares of Table I. Thus, if

$$M_1 = A,$$
$$M_2 = A + nB,$$
$$M_3 = A + nB + mC,$$

we may write, for instance,

$$z=\frac{C}{A+B+C}=\frac{n(M_3-M_2)}{(n-1)mM_1+(m-n)M_2+nM_3}.$$

In this case M_1, M_2, M_3 are independent and have zero covariances.

Consider now the simplified analysis of Table II.

TABLE II

Source of Variation	D.F.	Mean Square
Between classes . .	b	$A+nB$
Within classes . .	$a=(b+1)(n-1)$	A

For the ratio $z=B/(A+B)$ we derive, as before,

$$(A+B)^4S^2_z=A^2S^2_B+B^2S^2_A-2AB\,\widehat{\operatorname{cov}}\,(A,B)=A^2\left\{S^2_B+\frac{B^2}{A^2}S^2_A+\frac{2B}{A}\frac{S^2_A}{n}\right\},$$

giving, by substitution for S^2_A and S^2_B,

$$S^2_z=\frac{2A^2(A+nB)^2}{n^2(A+B)^4}\left\{\frac{1}{a}+\frac{1}{b}\right\}, \tag{2}$$

Formula (2) may be written as

$$S^2_z=\frac{2A^2(A+nB)^2}{(A+B)^4}\left\{\frac{n(b+1)-1}{n^2b(b+1)(n-1)}\right\},$$

or, approximately,

$$S^2_z=\frac{2A^2(A+nB)^2}{(A+B)^4n(n-1)b}. \tag{3}$$

The ratio z is of course an estimate of the intra-class correlation coefficient, the estimate of standard error by Fisher's formula being

$$S_z=\frac{\left\{1-\frac{B}{A+B}\right\}\left\{1+\frac{(n-1)B}{A+B}\right\}}{\sqrt{\frac{1}{2}n(n-1)(b+1)}}=\frac{\frac{A}{A+B}\cdot\frac{A+nB}{A+B}}{\sqrt{\frac{1}{2}n(n-1)(b+1)}},$$

with corresponding estimate of sampling variance

$$S^2_z=\frac{2A^2(A+nB)^2}{(A+B)^4n(n-1)(b+1)}$$

closely approximating formula (3).

SUMMARY

Inadequacies are pointed out of some existent methods of setting fiducial limits to estimates of heritability where more than two components of variance are involved. Although anormality of distribution may preclude the possibility of setting precise fiducial limits, a method is presented by which estimates of sampling variance may be derived for all ratios involving such components. For a simplified classification the method gives a close approximation to Fisher's standard error of the intraclass correlation coefficient.

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SUMMARY AND CONCLUSIONS

Part (1) of this thesis contains an account of developments in quantitative inheritance studies during the present century, with particular reference to poultry breeding. Current biometrical concepts and techniques are introduced by the method of path coefficients, and illustrations given of the correlations between relatives for autosomal and sex-linked inheritance, the contribution of environmental agencies, and the contribution of genetic and environmental agencies to correlations between different traits.

Estimation of the above correlations, and of heritability, is described in terms of analysis of variance and covariance, and the contribution of non-additive effects emphasised, with special reference to the detection of sex-linkage, maternal effects, dominance and epistasis. The fallacy of a recent suggestion for estimation of heritability, and its bearing on problems of sex-linked inheritance, is discussed.

Part (II) is an analysis of the variations of egg weight and its components, body weight, and age at sexual maturity, in the Brown Leghorn flock of the Poultry Research Centre, Edinburgh. Two supporting publications are included, the first dealing with preliminary studies on age at sexual maturity and the second with sampling errors of heritability estimates. The findings are summarised in sections as below:-

(1) Variations of Egg Weight.

Analysis of variance of March egg weight in a control line of Brown Leghorns indicates, in each of nine yearly samples, the primary dependence of individual variation on that of the male parent.

Chance sampling effects, non-additivity, non-randomisation of environment, inbreeding, relationship of the parents, and differences in genetic value of dams mated to particular sires are either insufficient

to account for the large sire components of variation or would operate in the reverse direction.

The apparent dependence of female offspring on sire variation suggests that sex-linked inheritance is operative and similar results implicit in the data from three other sources confirm this view. The contention is further strengthened by multiple regression analysis of the phenotypic values of offspring, dams and sires, the latter being scored on their mothers' performances. The degree of determination of offspring by paternal grandmother appears to be higher than determination by the dam itself, that by the maternal grandmother being negligibly small.

On the other hand the variations are small compared with a major difference in egg weight between two sub-populations. Comparison of reciprocal crosses between these two lines reveals no evidence of sex-linked variation. The results are consistent with the existence of genes of major and minor effect, small within-line sex-linked variations and major autosomal differences between lines. Either type of variation may, of course, be dependent on variations in other traits, such as body weight.

(2) Variations of Egg Weight Components

Analysis of total egg weight and weight of albumen, yolk, and shell, has been carried out for two samples. A discrepancy between them suggests firstly that the pattern of sex-linkage may be the result of reaction to a particular environment, the evidence of sex-linkage being less in birds subject to artificial lighting. Secondly the variations of total weight and albumen appear to follow the pattern set in yolk.

An interesting example of the ambiguity of isolated variance components is given in the case of shell

weight. A component suggesting maternal effects may be due to a type of epistatic variation whereby the response in shell weight to large increments in egg weight is not in proportion to the change for small increments.

(3) Variations of Body Weight and Egg Weight

Within-line analysis of variance of mature body weight reveals no obvious relationship of sex-linked variation of the two traits. Heritability is 0.77 with slight evidence of maternal effects or non-additivity, as judged by components of variance. Further analysis reveals a large covariance of sex-linked effects and it appears that the latter may be masked by non-additivity increasing the dam component of variance. The same observation follows from regression analysis; in accordance with expectation the intra-sire regression of offspring on dam gives a lower heritability of 0.45.

(4) Variations of Age at Sexual Maturity

(a) In each of three years studied the spring hatching range was divided into two sub-periods such that a number of matings were represented by offspring in each sub-period. Analysis of variance of age at sexual maturity reveals statistical interaction between family means and hatching period, the ranking of family means varying with date of hatch. A complete summary of this evidence of genotype-environment interaction is given on pages 74-75.

(b) The concept of heritability is discussed with reference to the tangible and non-randomised environmental influences operating on individual birds nearing sexual maturity, and it is suggested that genetic and environmental variance, as expressed in the usual

statistical formulation, may be largely synonymous terms. Estimation of heritability in different years, ranging from the sub-optimal conditions of war-years to conditions of 14-hour day-length, reveals marked differences in genetic and environmental variance and the reduction of both in the latter circumstances. In one group subject to 14-hour day length a comparison between early and late hatched pullets indicates that genetic variance is confined to the later hatched group and further substantiates the presence of genotype-environment interaction. A similar conclusion in the latter respect follows from family heterogeneity in regressions of age at maturity on date of hatch.

Except in the extreme of improved conditions, artificial lighting and early hatching, there is no marked reduction in heritability, and values in the region of 0.5 are obtained over all the years. It is, however, apparent that the expected increase of heritability with standardisation of environment is offset by the corresponding reduction of genetic variation.

There is no obvious relationship of sex-linked association of age at sexual maturity and egg weight, and variation of the former is again predominantly 'maternal'.

(5) A method of estimating the sampling variance of all heritability estimates derived from ratios of variance components is presented. A complete summary of this publication is given on page 96.

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(Separate reference lists for the supporting publications are given on pages 75 and 96).

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